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STUDIES ON THE OCCURRENCE, DISTRIBUTION AND PROGRESSION OF PERIODONTITIS

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PREFACE

This dissertation describes how knowledge and understanding of the occurrence, distribution and progression of periodontitis has advanced from the late 1970s to the present day, using the author's own investigations to illustrate how modern concepts have emerged.

The investigative work was begun in 1978, and comprised two studies: the first, a descriptive cross-sectional survey of 800 subjects; the second, a detailed 1-year longitudinal study of 11 patients.

This research gave rise, between 1984 and 1991, to five published reports which are re-typed as Chapters 2—6. Save for minor editing to conform to a common presentational style and reduce repetition, and the incorporation of a little additional material, Chapters 3—6 essentially reproduce the original publications. In writing Chapter 2, however, it was necessary to introduce a substantial amount of material not included in the original report, which was presented for publication in an abridged form.

In re-typing the five publications, no attempt has been made to update the textual references so as to portray the sequential acquisition of knowledge, untouched by hindsight.

The thesis begins with a review of the literature up to 1982, when the first report was submitted for publication, and ends with an overview of the investigations described in earlier chapters, thereby extending the review process to embrace more recent developments in this field.

Copies of the seven publications, which form the framework of this thesis, constitute its Appendix. Included with the five published original research reports, are two published literature reviews, comprising one journal article, submitted for publication in 1982, on which Chapter 1 is based, and a recent book chapter, which incorporates some of the material included in Chapter 7. These two literature reviews reveal how epidemiological concepts of periodontal disease have changed

between 1982 and 1995.

The seven publications are listed below in order, as they relate to the seven chapters:-

Jenkins, W.M.M. & Mason, W.N. (1984) Periodontitis in the United Kingdom: A literature review. *British Dental Journal*, **156**, 43—45.

Jenkins, W.M.M. & Mason, W.N. (1984) Radiographic assessment of periodontitis: A study of 800 unreferred patients. *British Dental Journal*, **156**, 170—174.

Jenkins, W.M.M. & Kinane, D.F. (1989) The 'high risk' group in periodontitis. *British Dental Journal*, **167**, 168—171.

Kinane, D.F., Jenkins, W.M.M., Adonogianaki, E. & Murray, G.D. (1991) Cross-sectional assessment of caries and periodontitis risk within the same subject. *Community Dentistry and Oral Epidemiology*, **19**, 78—81.

Jenkins, W.M.M., MacFarlane, T.W. & Gilmour, W.H. (1988) Longitudinal study of untreated periodontitis: (I). Clinical findings. *Journal of Clinical Periodontology*, **15**, 324—330.

MacFarlane, T.W., Jenkins, W.M.M., Gilmour, W.H., McCourtie, J. & McKenzie, D. (1988) Longitudinal study of untreated periodontitis: (II). Microbiological findings. *Journal of Clinical Periodontology*, **15**, 331—337.

Jenkins, W.M.M. & Allan, C.J. (1994) Classification and Epidemiology. In *Guide to Periodontics* (3rd edn), Ch 5, pp 41—50. Oxford: Wright.

Ethical approval of the experimental protocols of both the cross-sectional and longitudinal studies was granted by the Dental Ethics Committee of Greater Glasgow Health Board, and written informed consent was obtained from all participants.

ACKNOWLEDGEMENTS

In many ways, this project began in 1977 while I was on six-month secondment to the University of Gothenburg Dental School, Sweden. I am, therefore, greatly indebted to Professor J. Lindhe for sharing with me his ideas for better analysis of epidemiological data, and for suggesting the 'Björn ruler' as a suitable tool for a new cross-sectional radiographic survey of marginal bone loss.

It is with pleasure that I acknowledge both the assistance of Dr. W.N. Mason for providing the radiographic facilities, and the contribution of Mrs J. McNaught who took the radiographs.

Data processing for the cross-sectional study was performed by the Statistics Section of the Northern Regional Health Authority, for whose support I am extremely grateful. My thanks are also due to Mr W.H. Gilmour and Dr. G.D. Murray of the University of Glasgow for their help with further data processing and statistical analysis.

I gratefully acknowledge the assistance and cooperation of Dr. Eva Adonogianaki who identified and enumerated the carious and restored teeth, and Professor T.W. MacFarlane and his staff in the Microbiology Unit who carried out the bacteriological tests.

I would also like to express my sincere appreciation to Miss Pamela Gibb for typing the manuscripts.

Finally, I wish to thank Professor K.W. Stephen and Professor D.F. Kinane, without whose encouragement this dissertation would not have been written.

DECLARATION

The material contained in this thesis is original. The author planned the series of investigations, led the research and undertook all the clinical assessments, microbiological sampling and radiographic measurement of periodontal bone levels.

SUMMARY

The Periodontal Index of Russell (1956), either in its original or in modified form, was once the principal index for a series of epidemiological studies that correlated disease scores with a number of clinical and social determinants. By the late 1970s, however, there were considerable doubts about the validity and reliability of the Periodontal Index. There were substantial and unexplained differences in prevalence rates between different populations, and major inconsistencies were apparent in the reported prevalence of chronic periodontitis in the United Kingdom. It was also noteworthy that UK populations appeared to have substantially more periodontal disease than populations in the United States of America and Sweden.

It appeared that the diagnostic criteria, employed to describe prevalence and severity of periodontitis, often did not reflect the true presence or absence of the disease. Very few studies had been carried out which measured attachment loss directly by probing, and, although radiographic studies of bone loss were plentiful, most provided incomplete data, e.g. partial recording or one estimator of severity.

Most epidemiological data had been reported in mean severity scores, leading to the assumption that periodontitis was more or less evenly distributed among individuals of the same age and standard of oral hygiene, and, although it had been shown that the prevalence and severity of periodontitis increased with decreasing standards of oral hygiene, the data available were insufficient to indicate the extent to which unknown risk factors might influence the disease process.

Accordingly, central to the investigative work described in this thesis is a descriptive cross-sectional epidemiological study, based on a detailed radiographic analysis of periodontal marginal bone levels in 800 unreferred casual attenders at Glasgow Dental Hospital and School in 1978. The specific aims were to determine the prevalence, severity, extent and within-

population distribution of marginal bone loss, and to compare the findings with those obtained from a similar survey of Swedish shipyard employees (Björn, 1971).

A rotational tomographic view was taken for each patient, and the height of alveolar bone at each proximal surface was calculated with a transparent ruler using the crown tip and root apex as reference points. The ruler was calibrated to score bone loss in 'quarters' of optimum bone height (Björn & Holmberg, 1966).

A score value was awarded at 78.6% of proximal surfaces, the remainder being deemed unmeasurable. Marginal bone loss increased with age and was slightly more severe in males than females and in males among the lower social classes. The development of bone loss with age was attributable both to an increase in affected surfaces and to progressive destruction at some of these surfaces.

A high prevalence of marginal bone loss was found. However, very few individuals had *generalised* bone loss exceeding 25% of optimum bone height. Furthermore, only a tiny proportion of *teeth* were affected by bone loss exceeding 50% of optimum height.

Use of the 'Björn ruler' enabled the findings for males aged 20—64 years to be viewed alongside those of a comparable population of Swedish males. These different populations had similar marginal bone levels.

Further analysis revealed that, with age, in parallel with other investigations, a small but increasing minority of patients accounted for most of the bone destruction. For example, in the 50—73-year age-group, 28% of patients accounted for 75% of the advanced bone loss.

This cross-sectional study showed, in common with established belief, that age, sex and socio-economic status are determinants of periodontitis. However, such great variation existed within these subgroups that none of these attributes could be considered to be reliable indicators of the presence of periodontitis or predictors of its progress. The study was, therefore, extended to include a radiographic assessment of

carious and restored teeth among the 800 subjects so that the susceptibility to caries and periodontitis within the same individual could be compared. The caries risk was determined radiographically from the total decayed and filled teeth (DFT), as a percentage of all the teeth measured. The Mantel-Haenszel technique was used for analysis of the relationship between periodontitis and caries, and data were stratified on four categories of age, sex and numbers of teeth present. This analysis failed to reveal any systematic patterns, indicating that the risks of caries and periodontal disease are unrelated ($\chi^2 = 0.00$; $df = 1$; $p > 0.50$). In addition, a regression analysis, controlled for sex and age, indicated a marked lack of association between caries and periodontitis ($p = 0.94$). Thus, although these common diseases share putative aetiological factors such as poor oral hygiene practices, the major risk factors are probably quite different.

In a further attempt to determine what characteristics are peculiar to the susceptible minority of individuals, a longitudinal clinical study was undertaken. Its principal aim was to investigate the use of certain clinical and microbiological criteria to predict periodontal breakdown during a 1-year period. A further aim was to establish whether the act of collecting subgingival plaque samples periodically throughout the observation period would have any effect on the clinical or microbiological variables. The study population comprised 11 volunteers (aged 32—51 years) with persistent advanced periodontitis and inadequate plaque control in spite of a previous intensive course of hygiene therapy. From the left jaw quadrants, 89 teeth were selected, yielding 148 bleeding pockets of ≥ 4 mm depth. From the right jaw quadrants, 74 teeth were selected, yielding 117 bleeding pockets of ≥ 4 mm depth. All subjects were examined on seven occasions at 2-month intervals when plaque index scores, dichotomous measurements of gingival redness, pocket depths and attachment levels were recorded. Bacteriological sampling was carried out at each visit for each site only in the left jaw quadrants, while the right jaw quadrants were sampled only at

the first and last visits. During the study, no subgingival instrumentation was performed, except at three sites which exhibited progressive loss of attachment of 3 mm. These teeth were withdrawn from the study for ethical reasons. On completion of the study, the sequential changes in probing attachment level at each site were subjected to regression analysis to determine the direction and extent of attachment change which had taken place at each site. Further analysis revealed that, neither the plaque index scores, the presence of gingival redness, nor the pocket depth measurements could be used to predict attachment change.

The microbiological markers investigated were the percentage spirochaetes and percentage black-pigmented *Bacteroides* species. Possible correlations between these microbial variables and attachment changes were investigated at baseline, at 12 months, and by using the microbial data accumulated over all seven visits. Significant differences were observed only at the 12-month visit when the percentage spirochaetes of both test and control sites were significantly lower in subjects showing the greatest improvement in attachment level. Overall, these results indicate that quantification of either spirochaetes or black-pigmented *Bacteroides* species cannot be used reliably to identify or predict disease-active sites.

Comparison of right and left jaw quadrants showed that the act of sampling at 2-month intervals had no effect on either the clinical or microbiological variables.

In conclusion, these cross-sectional epidemiological and longitudinal clinical studies have demonstrated the frequent occurrence and complex distribution of periodontitis, the existence of a 'high risk' group and the failure of certain associated clinical and microbiological variables to predict its progress in time for preventive action to be taken.

CHAPTER 1

HISTORICAL INTRODUCTION

1.1 Introduction

This chapter reviews the epidemiology of periodontitis according to the knowledge available by 1982 when the first part of the present investigative work was completed.

Systematic epidemiological study of periodontitis began in the 1950s and, within a few years, a concept of the nature of periodontal disease had been established which was to survive until the late 1970s. It was considered that periodontal disease was widespread and showed a continuous progression with age: gingivitis, if left untreated, increased in extent and severity until periodontitis supervened; all individuals were susceptible to periodontitis, especially if oral hygiene was poor; the severity increased with age resulting eventually in tooth loss; and tooth mortality studies supported the belief that periodontal disease was a major cause of tooth loss (Scherp, 1964; Waerhaug, 1966).

During the 1970s, however, this orthodoxy was increasingly challenged, and it became apparent that the philosophical base for much of the epidemiological research was faulty. In the critical analysis of epidemiological research which follows, the data used for illustrative purposes are, wherever possible, those obtained in the United Kingdom.

1.2 The Periodontal Index (PI)

The PI, introduced by Russell (1956), was used extensively for over two decades. This index was based on the assumption that gingivitis and periodontitis represented a disease continuum, the former increasing in extent and severity until periodontitis developed. Thus, a non-linear weighted scale was used to combine gingivitis and periodontitis as illustrated in Table 1.1. The tissues of each tooth were examined and assigned a numerical value as shown in Table 1.1. To arrive at a final score, the values for the individual teeth were summed and divided by the number of teeth examined. Russell (1967) suggested that these mean score values were indicative of various clinical diagnoses (Table 1.2). A population mean score could be obtained by taking an average of individual scores.

1.2.1 PI surveys

The PI was used in the late 1960s in three large surveys of United Kingdom populations. Swallow & Adams (1967) set out to examine all adults within two defined areas in the Rhondda Valley of South Wales. Sheiham (1969) obtained his sample by random selection from the lists of employees of industrial units, two in London and one in Warrington. Sheiham & Dimmer (1971) used a random sample of white and blue collar workers at a large Belfast factory. The findings of all three studies were similar, but those of Sheiham (1969) were reported in most detail: The PI increased from 1.23 for 15—29-year-olds to 5.8

Table 1.1 Scoring Criteria for the Periodontal Index (after Russell, 1956)

Score Value	Criterion
0	No overt gingivitis or loss of function
1	Overt gingivitis not circumscribing the tooth
2	Circumscribing gingivitis
6	Early periodontitis (gingivitis with loss of attachment)
8	Advanced periodontitis with impaired function

Table 1.2 Relationship between subject mean Periodontal Index scores and the overall clinical diagnosis (after Russell, 1967)

Diagnosis	Score range
Clinically normal	0 - 0.2
Gingivitis	0.3 - 0.9
Borderline periodontitis	0.7 - 1.9
Established periodontitis	1.6 - 5.0
Terminal periodontitis	3.8 - 8.0

for 60—65-year-olds. According to the criteria set out in Table 1.2 (Russell, 1967), this meant that the 15—29-year-olds had 'borderline periodontitis' and the 60—65-year-olds 'terminal periodontitis'. By those same criteria, 42% of the total sample were in the terminal stages of periodontal disease and about to lose large numbers of teeth. Another important observation was the contrast in PI scores between this population and a comparable population of white Americans (Kelly & Van Kirk 1965): when British and American persons of the same age range were compared, British persons had a mean PI, three times higher than the Americans. Even allowing for the better standard of oral hygiene achieved by the Americans, it is questionable whether differences of this magnitude could be real, and indeed, Sheiham conceded that examiner variability in use of the PI may have accounted for some of the difference.

Comparison of other populations revealed similar inconsistencies. Thus, while early literature reviews described a close correlation between oral hygiene and periodontal disease (Scherp, 1964; Waerhaug ,1966), this could not always be confirmed. For example, a United States population (Johnson, Kelly & Van Kirk, 1965) and Ecuador and Montana populations (Greene, 1963) had similar age-specific PI scores, but the latter much higher oral hygiene scores. Similarly, Nigerian Yorubas (Sheiham, 1967) had similar PI values to those of the aforementioned United Kingdom population (Sheiham, 1969) but a much poorer standard of oral hygiene. These findings suggested either: that oral hygiene and periodontal disease were not well correlated; that

susceptibility to periodontal disease varied substantially between populations; or that the reproducibility of the oral hygiene index and/or the PI were poor.

1.2.2 Shortcomings of the PI

In due course, the PI system was found to possess several serious deficiencies.

Firstly, the criteria used to diagnose periodontitis were open to question. Periodontitis (true pocketing) was originally detected, not with a periodontal probe, but using a blast of air from a chip syringe. Flaccid, inflamed gingiva would be easily displaced from the tooth surface yielding a diagnosis of periodontitis if the epithelial attachment was judged by this procedure to be broken. On the other hand, fibrotic gingiva would resist displacement, even if pockets were present. In some of the later PI surveys (e.g. Sheiham, 1969) this difficulty was apparently overcome by using a periodontal probe and diagnosing periodontitis when pockets of 3 mm or more were detected. This may help to explain why these surveys gave rise generally to higher PI scores.

The lack of objective scoring criteria applied not only to the diagnosis of early periodontitis, as described above, but also to terminal periodontitis (score value 8) the presence of which was determined by the demonstration of mobility, migration, a dull percussion note or depression of the tooth within its socket.

Thus, there were good theoretical grounds for doubting the validity and reliability of the PI. However, its most serious

shortcoming was the philosophy underpinning composite indices that regarded gingivitis and periodontitis as merely two stages in a single disease process, in spite of the fact that cross-sectional investigations had failed to establish any relationship between gingivitis and alveolar bone loss (Massler, Mühlemann & Schour, 1953; Rateitschak, Marthaler & Engelberger, 1964). During the 1970s, further reports cast serious doubts on this hypothesis. Glass *et al.* (1973), for example, evaluated six periodontal disease variables in 635 men, finding that gingivitis and periodontitis were poorly correlated. Lindhe, Hamp & Löe (1973) found that in-bred dogs with identical housing and diet, allowed to accumulate plaque for four years, all developed gingivitis, but 20% of the animals failed to develop periodontitis. Although gingivitis developed around most teeth, loss of attachment predominantly affected the premolars. The lack of progression from gingivitis to periodontitis was apparent not only in young dogs, but also among the older dogs. Thus, some dogs and some sites were clearly resistant to periodontitis in spite of being affected by gingivitis. Further evidence that gingivitis and periodontitis were not continuous phenomena was presented by Page & Schroeder (1976) in their classic paper on the histopathological features of periodontal diseases.

1.3 Tooth mortality studies

If severe periodontal disease was highly prevalent, as suggested by many PI studies, this should be reflected in tooth

mortality statistics. Yet, in spite of recording very high PI values in a Nigerian population, Sheiham (1967) noted that the number of teeth missing in all age groups was extremely small. There were, furthermore, wide variations in the reasons reported for tooth extraction, even in industrialised countries sharing similar standards of oral hygiene and levels of dental care. For example, it was reported that the percentages of all teeth extracted for periodontal reasons in Sweden (Lundqvist, 1967) and Denmark (Gad & Bay, 1972) were 11% and 30% respectively.

In due course, doubts were expressed about the validity of tooth mortality studies. Todd & Whitworth (1974) pointed out that dentists' and patients' attitudes, together with technical difficulties associated with provision of treatment may be significant factors determining the timing of extraction which obscured the significance of caries and periodontal disease.

It was also difficult to reconcile cross-sectional tooth mortality data, which suggested high rates of tooth loss from periodontal disease, with longitudinal studies which showed that, in untreated individuals up to approximately 40 years-of-age, the mean rate of attachment loss was approximately 0.1 mm per year (Suomi *et al.*, 1971).

1.4 The UK national surveys of adult dental health

The published record of periodontal disease in the United Kingdom includes the reports of the national surveys of adult

dental health in England and Wales in 1968 (Gray *et al.*, 1970), in Scotland in 1972 (Todd & Whitworth, 1974) and in the United Kingdom in 1978 (Todd, Walker & Dodd, 1982). For each national survey, subjects were selected from the electoral register, with a supplementary sample of young persons, to give a total sample which was as representative as possible of the population in the age-range 16 years-and-over. In order to complete each survey within a reasonable time, it was necessary to recruit a large number of examiners since the subjects were widely dispersed. Not all examiners were experienced in the examination of periodontal disease and, although each survey was preceded by a period of intensive training, it was necessary to use rather crude indices of disease status in an attempt to reduce individual examiner variation to an acceptable level. Furthermore, to achieve a high response rate, the dental examinations were conducted in the subjects' homes, thus limiting the scope of the examination. It was acknowledged that these constraints would result in underestimation of true disease levels.

All three national surveys used similar criteria for detection of periodontitis, which was diagnosed when pocket depths either reached or exceeded a threshold level of 3 mm. This strategy, also used in some PI surveys, was based on the assumption that, in the normal healthy periodontium, probing depths rarely exceed 1—2 mm. It was, therefore, thought reasonable to assume that pockets ≥ 3 mm in depth might be a sign of disease. However, false positive results could be obtained in cases of gingival hyperplasia and false negative

findings would arise if recession had kept pace with loss of attachment.

1.4.1 England and Wales 1968

The 1968 England and Wales survey limited its assessment of 'periodontitis' to *enumerating* pockets of ≥ 3 mm depth, and found that they affected only two teeth per person, on average. By contrast, using the same examination criteria, Sheiham & Dimmer (1971) found that there were, on average, 10.2 pockets per person among a Northern Ireland factory population. The disparity in the results of these two surveys is difficult to explain. In the Northern Ireland study, however, males outnumbered females by 5 to 1, and this may account to a small extent for the higher figure in that study, since periodontal conditions are known to be worse in males than in females (Waerhaug, 1966).

The 1968 England and Wales survey report also gave details of the distribution of gum conditions around the mouth, from which it may be calculated that, for adults of all ages, only 7.2% of first molars exhibited chronic periodontitis, as identified by pockets of ≥ 3 mm depth.

1.4.2 Scotland 1972

The proportion of first molar teeth affected by periodontitis in England and Wales was slightly higher than that reported in the 1972 survey of Scottish adults, where only 2% of first molars were judged to be affected by chronic periodontitis, according to the same 3 mm pocket depth criterion. This might

suggest that periodontal disease was not a significant problem in Scotland, since pocket formation does not usually occur in other regions of the dentition without including one or more of the first molars (Hugoson & Rylander, 1982). In the Scottish survey, however, pockets were not probed unless the buccal or lingual marginal gingiva was 'markedly red or bluish red in contrast to the colour (usually pink) of adjacent healthy tissue'. Minor inflammatory changes were presumably to be ignored, and inevitably, a large number of pockets would be missed since it has long been known that periodontitis is not always accompanied by readily detectable gingival changes (Marshall-Day, Stephens & Quigley, 1955). The examiners, once they had identified marked gingivitis in buccal or lingual marginal gingiva, were instructed to confine their probing to that part of the tooth circumference and not to look for pockets in the papillary area. Since the frequency of pockets of ≥ 4 mm depth on proximal surfaces greatly exceeds that on buccal or lingual surfaces (Hugoson & Koch, 1979), failure to probe proximal surfaces would lead to further underestimation of disease prevalence.

1.4.3 United Kingdom 1978

In the 1978 United Kingdom survey, periodontitis was diagnosed when tooth mobility or pockets of > 3 mm depth were detected. Thus, periodontitis was reported to affect 3% of 16—24-year-olds, and to increase with age to affect 65% of over-55-year-olds. This contrasts markedly with the earlier study of Sheiham (1969) who found that the prevalence of pocketing was

50.5% for 15—19-year-olds, 75.9% for 20—24-year-olds and 100% for 55—59-year-olds. This huge difference, however, can readily be explained by reviewing the respective examination criteria. Although both studies used similar criteria to diagnose chronic periodontitis, i.e. a pocket depth threshold of 3 mm, teeth in the 1978 UK survey, like those in the earlier Scottish survey, were investigated for periodontitis by probing only if they exhibited intense gingivitis or a marked change in gingival contour. Sheiham's data, furthermore, were in line with the 31% prevalence rate for pockets of > 3 mm depth, reported by Downer (1970) from a small survey of periodontal conditions in 11—14-year-old girls in an English school.

1.5 Attachment level surveys

Although the detection of attachment loss in most epidemiological studies was based on ease of deflection of the gingiva, or on pocket depth assessment, by 1982 a few studies had been published which relied on direct measurement of the clinical attachment level, a more time-consuming procedure requiring more skill and precision. Attachment level measurements were first advocated by Ramfjord (1959) and included in his Periodontal Disease Index, a composite index which merged gingivitis with attachment loss scores and which, therefore, suffered from many of the drawbacks already described for the PI.

Attachment level measurements were first used in a United

Kingdom survey by Lennon & Davies (1974), who examined the incisors and first molars of 15-year-old children and reported prevalence rates of 46% and 11% for thresholds of attachment loss ≥ 1 mm and ≥ 2 mm, respectively. Later, Mann *et al.* (1981) reported similar findings in an adolescent population in the United States of America, but Hoover, Ellegaard & Attström (1981) detected a somewhat lower prevalence of attachment loss in Danish adolescents.

1.6 Radiographic diagnosis

While the destructive effect of periodontitis can be assessed clinically only by measuring attachment levels, it can also be detected radiographically by measuring reductions in alveolar bone height.

Only interdental bone may be visualised; facial and lingual bone is almost completely obscured by the radio-density of the superimposed tooth. On the other hand, it is well established that the main clinical manifestations of periodontal disease are observed most frequently on proximal tooth surfaces (Hugoson & Koch, 1979; Becker, Berg & Becker, 1979), and that attachment loss proceeds faster proximally than facially or lingually (Becker *et al.*, 1979). Perhaps a more serious weakness, inherent in the radiographic assessment of bone loss is the tendency to overestimate the extent of bony support when an interproximal crater is present, overshadowed by facial and lingual bone margins.

Epidemiological assessment of marginal bone has been based on apparent qualitative changes as well as on reduction of alveolar bone height. The traditional radiographic signs of early periodontitis, prior to reduction in crestal bone height, are marginal widening of the periodontal membrane space and loss of surface continuity of the alveolar crest.

1.6.1 Bitewing radiography in adolescents

Hull, Hillam & Beal (1975) and Davies, Downer & Lennon (1978) carried out bitewing radiographic investigations of 14-year-old English schoolchildren to diagnose marginal bone loss. Their criteria for bone loss included widening of the periodontal ligament space, irregularity of the alveolar crest or a bone level beyond a threshold value of 3 mm between the cemento-enamel junction and the alveolar crest. The prevalence rates for bone loss, as reported in those studies, were 51.5% and 44.0% respectively. However, Blankenstein, Murray & Lind (1978) in a study of bitewing radiographs from 1645 English and Danish school children in a 13—15-year age-group, found only one child with an alveolar crest situated at a distance from the cemento-enamel junction of > 3 mm. This would suggest that the high prevalence rates reported by Hull *et al.* (1975) and Davies *et al.* (1978) may be attributable to qualitative changes rather than an alveolar bone level of > 3 mm. Later, the significance of apparent qualitative changes in the radiographic image of alveolar bone was questioned by Greenstein *et al.* (1981), who showed that the integrity of the

crestal lamina dura may be unrelated to visible inflammation, bleeding on probing, pockets or loss of attachment.

1.6.2 Full mouth radiography in adults

Outwith the United Kingdom, by 1982, several studies had already been carried out on large populations of all ages, using full mouth intra-oral radiographs, so that prevalence and severity could be assessed for the whole dentition. Among those studies in which the diagnostic criteria were reported in some detail was a survey of 1279 subjects in the area of Boston, USA (Marshall-Day *et al.*, 1955). An alveolar bone level of 2 mm was used as the threshold for bone loss and severity was measured on a scale of 1—10, where 10 was bone loss extending to the root apices. Thus, Marshall-Day *et al.* (1955) reported a prevalence of 81% by the age of 31—34 years, rising to 95% by the age of 52—55 years. Severity data were described in age-specific severity scores showing that, by 35—39 years, average bone loss had amounted to approximately one third of the total root length and, in the 60—65-year age range, more than half of the total root length was involved.

Many other radiographic surveys were carried out. These are noteworthy, mainly for confirming that periodontitis was more prevalent and more severe in individuals of increasing age and with decreasing levels of oral hygiene (e.g. Belting, Massler & Schour, 1953; Schei *et al.*, 1959; Björn, 1971).

1.7 Data analysis

It will be apparent from the foregoing account of periodontitis that no clear picture could be drawn of the occurrence and distribution of periodontitis prior to 1982. The confusion may be attributed to the use of many different diagnostic criteria, some of which were of questionable validity. It was, therefore, difficult to compare the variation in disease experience *between* populations. A further hindrance to the understanding of periodontitis was the practice in most epidemiological studies of reporting the occurrence and distribution of periodontal disease, either in terms of mean severity scores, or by providing only one estimator of extent or severity. Thus, knowledge of the distribution of periodontitis *within* populations (i.e. within and between individuals) was lacking: for example, although periodontal conditions were known to worsen with increasing age, it was often impossible to determine from published data whether this was due to an increase in the number of teeth or individuals affected, to small increments in disease severity affecting a large proportion of teeth or individuals, or to marked deterioration in a small proportion of teeth or individuals. The 1972 survey of Scottish adults (Todd & Whitworth, 1974) was one of the first to recognise this shortcoming of most previous reports by presenting frequency distributions which described the percentages of teeth affected by different levels of disease severity. Unfortunately, as previously noted, inappropriate diagnostic criteria were used.

Surprisingly, several years passed before this method of reporting periodontal disease data was adopted again. Then, in 1982, in a single issue of *Community Dentistry and Oral Epidemiology*, two papers were published which challenged the traditional view of the epidemiology of periodontal disease. Hugoson & Jordan (1982) examined 600 randomly selected subjects in Sweden, placing each individual in one of five categories of disease severity according to the predominant periodontal diagnosis made from a full oral examination. Only a small minority of individuals had severe generalised disease, even though gingivitis and attachment loss were highly prevalent. Cutress, Powell & Ball (1982), reporting the findings of a survey of two South Pacific Island populations, observed that, even where plaque accumulation and gingivitis were endemic, only a small proportion of individuals had developed alveolar bone loss of sufficient severity to cause major periodontal breakdown and multiple tooth loss.

1.8 Conclusions

By 1982, the following conclusions on the epidemiology of periodontal disease could be drawn:

1. The diagnostic criteria used to describe prevalence and severity of periodontitis often did not reflect the true presence or absence of the disease.
2. The prevalence and severity of periodontitis increased

with increasing age and decreasing oral hygiene. However, the data available were insufficient to indicate the extent to which unknown risk factors might influence the disease process.

3. Different examiners, using similar or different criteria in comparable populations (e.g. United Kingdom populations), had produced widely varying estimates of disease prevalence and severity.
4. Only a few studies had been carried out which measured attachment loss directly by probing.
5. Attachment loss studies in teenage children suggested that the prevalence of attachment loss was high. Very few studies, using attachment loss measurements, had been carried out in adults.
6. Radiographic studies of bone loss were plentiful but most provided incomplete data, e.g. partial recording or one estimator of severity.
7. Most epidemiological data had been reported in mean severity scores. It was, therefore, assumed that periodontitis was more or less evenly distributed among individuals of the same age and standard of oral hygiene.
8. Although most reports of tooth mortality cited periodontal disease as a major cause of tooth loss, the criteria used to determine the reasons for extraction in these studies had been questioned.
9. Two studies had reported that severe disease may be concentrated in a few individuals.

1.9 Aims

The *general* aims of the present series of investigations, to be described in more detail in forthcoming chapters, were:

1. To determine the occurrence and distribution of periodontitis from a large cross-sectional radiographic study of a United Kingdom population using validated diagnostic criteria, and, by group comparisons, to look for risk markers or disease determinants.
2. To assess basic clinical and microbiological variables as risk markers of progressive periodontitis from a detailed longitudinal clinical study of a small group of untreated periodontal patients.

CHAPTER 2

RADIOGRAPHIC ASSESSMENT OF PERIODONTITIS: A STUDY OF 800 UNREFERRED PATIENTS

2.1 Introduction

This chapter describes the rationale of radiographic methods of periodontal diagnosis in prevalence studies, and reports the findings of a radiographic investigation involving 800 adults from a population of dental hospital out-patients in Glasgow.

2.1.1 Radiographic bone-height determinations

It has been shown both histologically (Gargiulo, Wentz & Orban, 1961; Waerhaug, 1979) and radiographically (Herulf, 1950; Zachrisson & Alnaes, 1973) that the alveolar bone margin of normal, intact periodontal tissues lies 1 mm apical to the cemento-enamel junction. Radiographic measurements of reduction in alveolar bone height from this norm (the optimum bone height) have been used to determine the prevalence and severity of marginal bone loss (Schei *et al.*, 1959).

Quantitative measurement of marginal bone loss involves a degree of objectivity lacking in clinical indices and qualitative radiographic evaluation. Epidemiological assessment of the amount of alveolar bone loss has been undertaken by direct millimetric measurement (Hull *et al.*, 1975; Davies *et al.*, 1978) and by a proportional principle

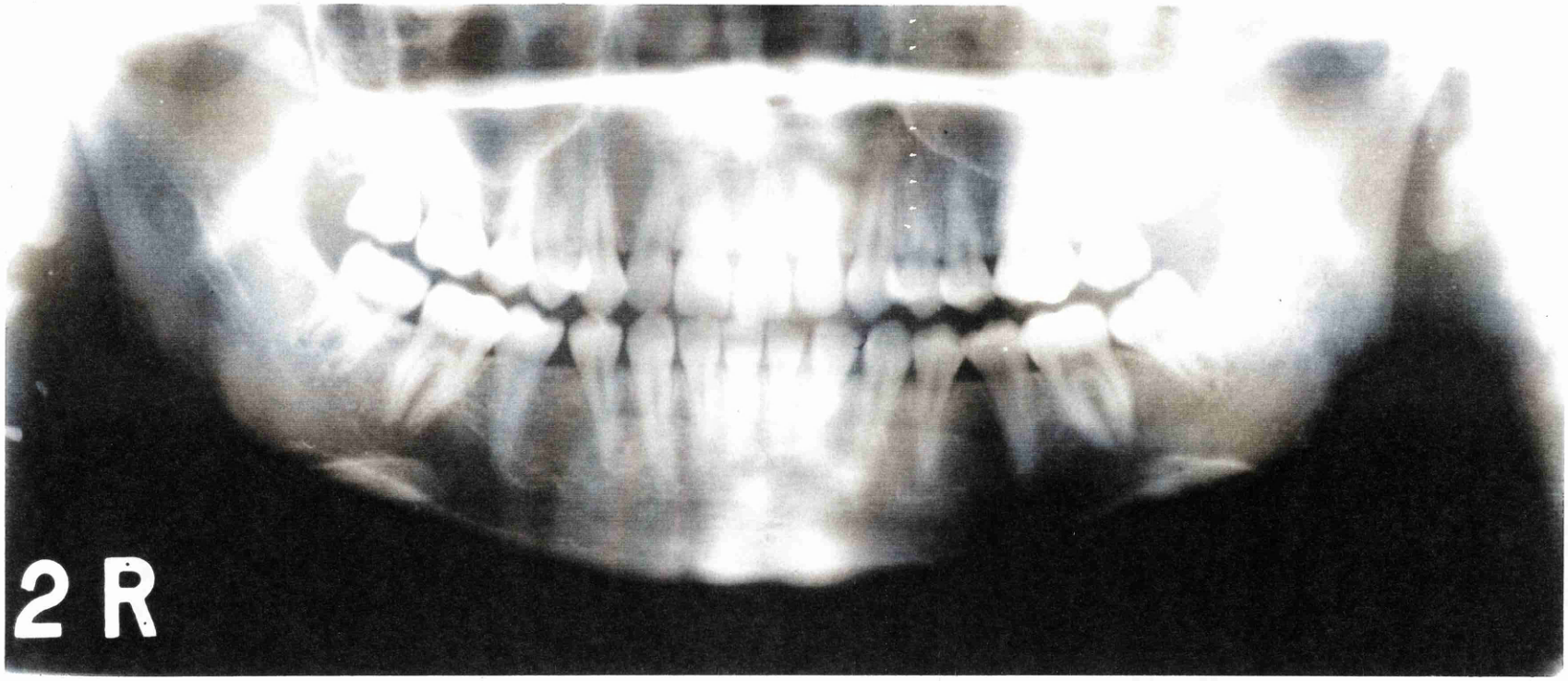
(Marshall-Day *et al.*, 1955; Schei *et al.*, 1959; Helminen-Pakkala, 1968), relating the actual alveolar bone level to the optimum bone height.

2.1.2 Radiographic techniques

For periodontal diagnosis in clinical practice, the radiographic technique of choice is the long-cone paralleling method, which produces a life-size image of the tooth with excellent definition. Unfortunately, this technique is time-consuming and costly, and is, therefore, inappropriate for epidemiological work. Panoramic radiography, by which the entire dentition may be examined with one exposure, is a simple, comfortable and comparatively inexpensive alternative, which subjects the study population to minimal radiation. Methodological studies have shown that, for quantitative assessment of marginal bone loss, orthopantomograph (OPG) radiographs (Figure 2.1) compare reasonably well with intra-oral radiographs. Björn & Holmberg (1966), for example, assessing bone levels as a percentage of total tooth length, obtained good agreement between intra-oral and OPG radiographs with respect to subject mean bone scores. Ainamo & Tammisalo (1967) showed that OPG radiographs yielded bone loss measurements which deviated less from the actual measurements than the results obtained from intra-oral, bisecting-angle radiographs. In a further investigation, these authors observed that, although detection of marginal bone loss by intra-oral radiographs was more reliable in the upper premolar region, or when bone loss involved the cortical layers or the

Figure 2.1

Orthopantomograph radiograph with no evidence of marginal bone loss. Note the typical overlapping of maxillary canines and premolars.



upper furcation sites, OPG radiographs were superior when interdental cancellous bone was involved or when lesions were very advanced (Ainamo & Tammisalo, 1968). Gröndahl, Jönsson & Lindahl (1971) showed that, compared to intra-oral films, obtained by the long-cone paralleling technique, OPG radiographs appeared to underdiagnose early marginal bone loss. Most significantly, blurring, low contrast and overlapping (Figure 2.1) reduce the number of surfaces which can be measured on OPG radiographs, compared to intra-oral radiography, which permits bone level measurements on virtually every proximal surface. This deficiency of the panoramic technique, although limiting its usefulness in clinical work, was nevertheless considered acceptable in epidemiological research (Björn & Holmberg, 1966).

2.1.3 Aims

The purpose of this survey was to obtain radiographic data on the prevalence and severity of marginal bone loss among casual attenders at Glasgow Dental Hospital and School. The survey method and statistical treatment of the results were selected to address the shortcomings of previous epidemiological reports, as identified in the literature review of Chapter 1.

The specific aims were to determine the prevalence and severity of marginal bone loss in a population of dental out-patients in Scotland; to ascertain the within-population distribution of marginal bone loss; and to compare the findings with those obtained from a similar survey of Swedish shipyard employees (Björn, 1971).

2.2 Material and methods

2.2.1 The sample

The study population comprised 800 unreferred dentate individuals of 16 years-and-over who presented as 'casual' patients for examination at Glasgow Dental Hospital and School during the period: February to May 1978. Pregnant women were excluded.

Individuals were questioned as they assembled to await diagnosis. Those who had at least one fully erupted tooth remaining were told of the purpose of the survey and invited to participate. The refusal rate was 1.0%. Written informed consent was obtained from those who agreed to participate. The age, sex and occupation of each participant was recorded. No clinical examination was carried out, but each participant's record was later traced to determine how his or her treatment needs had been met.

The protocol for this study was approved by the Dental Ethics Committee of Greater Glasgow Health Board.

2.2.2 Radiographic procedures

A rotational tomographic view (Figure 2.1) was taken for each patient using the Siemens Orthoceph 3. Kodak X-Omat RP film (15 x 30 cm) in a Siemens Palomex cassette, with super-high-speed intensifying screens, was used at operating factors of 15 mA and 65—75 kV to record the images. Each radiograph was then duplicated for retention in the patients' hospital records, and to permit repeat evaluations to be made for assessment of examiner variability.

2.2.3 Measurement technique

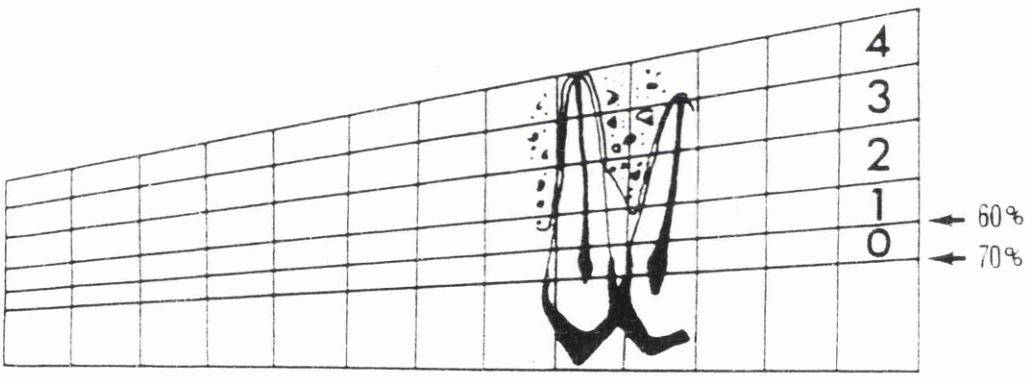
All radiographs were assessed and data collected by one individual (the author). The technique of radiographic assessment was similar to the method of Björn & Holmberg (1966) who, because the cemento-enamel junction is frequently not visible on OPG radiographs, designed a transparent ruler to calculate the height of the alveolar bone in proportion to the total length of the tooth. The ruler was calibrated so that bone loss could be scored in terms of 'quarters' of optimum bone height, which, according to the investigation of Engelberger, Rateitschak & Marthaler (1963), was considered to be 65 ± 5% of the total tooth length. However, to allow for variations in crown : root ratio and to avoid over-estimating the frequency of disease, Björn & Holmberg (1966) considered bone loss to be absent unless the bone level was less than 60% of total tooth length (Figure 2.2).

The validity of these criteria of bone loss is supported by the work of Hugoson & Koch (1979) who showed radiographically that the average bone height among 100 15-year-olds (among whom one would not expect to find widespread evidence of bone loss) was 63.6% of the total tooth length.

To test this criterion further for the present study, proximal surface measurements were made of 320 extracted teeth (20 of each tooth type). The distances from crown tip to root apex and from cemento-enamel junction to root apex were measured. The latter distance was reduced by 1 mm in each case to obtain the normal bone height, and, thereby, the amount of tooth normally invested in bone was calculated as a percentage

Figure 2.2

Ruler for measuring bone loss. Calibration of ruler into four grades of bone loss. Grades 2, 3 and 4 are based on an optimum bone height of 65% while Grade 1, denoting the early stage of bone loss, is based on an optimum height of 60%, so as to avoid including borderline cases among pathological ones. Where no bone loss has occurred the alveolar crest should lie within the 'zero' band. The tooth illustrated gives a proximal surface score of Grade 1 on one surface and Grade 2 on the other.



of total tooth length. Thus, the 'optimum' bone height in this study was 64.4% ± 3.78 (SD) of tooth length. Only 11.4% of proximal surfaces, because of relatively high crown : root ratios, appeared to have been invested by bone to less than 60% of tooth length. This variation, however, meant that the ruler could not be relied upon in every case to record bone loss accurately when crown tip and root apex reference points were used, making adjustments necessary at a minority of surfaces (see below).

All identifiable alveolar bone margins on mesial and distal surfaces of all teeth were marked with a felt-tip pen together with their crown tips and root apices. In the case of the multi-rooted maxillary molars, the buccal root tips were marked. Each proximal surface was assessed by applying the ruler over the radiograph (Figure 2.2) with its coronal and apical baselines coincident with the crown tip and root apex respectively. The ruler was inclined so that the long axis of the tooth was parallel to a line perpendicular to the coronal baseline. After confirming that the cemento-enamel junction, if visible, lay within the zero bone score band, the bone loss score for each proximal surface was obtained according to the position of the bone margin within the ruler grid system. Where a line was located exactly over the bone margin reference mark, the lower score was recorded.

Marginal bone loss in these cases was, therefore, assumed to be present when the alveolar bone height was less than 60% of total tooth length (Figure 2.2). If bone loss was present according to that criterion, it was graded on a severity scale

of 1—4 representing bone loss in four quarters of optimum bone height (65% of total tooth length). If bone loss was absent, a score of '0' was given.

Although crown tips and root apices were used as reference points in preference to the frequently unidentifiable cemento-enamel junction, the latter was used, when visible, to assess the positioning of the ruler. In some cases, where the anatomical crown : root ratios were high, the cemento-enamel junction was itself located apical to the zero bone score band. As a precaution against over-estimating bone loss when such teeth were identified, the ruler was adjusted to place the cemento-enamel junction just within this band, to reflect more accurately the actual proportional loss of bone, if any. In these cases, of course, the cemento-enamel junction replaced the crown tip as a reference point.

Conversely, in cases of low crown : root ratio, if the cemento-enamel junction was identified coronal to the zero bone score band, the ruler position was adjusted to bring it just within this zone.

A record of non-measurable surfaces was also kept.

Once all the original 800 radiographs had been measured, intra-examiner variability was assessed by repetition of the marking and measurement procedure on 80 radiographs (10% of the total) selected by random numbering from the collection of 800 unmarked duplicate radiographs.

2.2.4 Computational Procedures

Tooth surfaces, scored as unmeasurable, were treated

statistically as though no bone loss was present, and counted with surfaces which received bone loss scores of 'zero'.

The data were analysed in two ways:

(a) Mean bone loss scores for individuals and population subgroups were obtained by summation of bone loss scores 0—4 and division by the number of surfaces.

(b) A 'tooth' score was obtained by selecting the greater of the two proximal surface scores. Thus, a tooth, scoring '1' for mesial bone loss and '2' for distal bone loss, received a score of '2'. The tooth scores, therefore, represented the greater amount of bone loss identifiable on radiograph for each particular tooth. These data were then grouped according to severity, and a series of frequency distributions produced.

2.3 Results

2.3.1 Age, sex and number of teeth present

Table 2.1 shows the age and sex distribution, with the average number of teeth present in each age-group. Males formed 60.6% of the sample and out-numbered females in each age-group, except in the 16—19-year age-group. The modal age-group was 20—24 years for males and 16—19 years for females; 23.7% of males and 27.3% of females fell within these age ranges. Out of the 155 individuals in the 16—19-year age-group, only 19 were 16 years-of-age. Of the 108 individuals in the 45—73-year age-group, only 11 were between 60 and 73 years-old.

The average number of teeth present fell from 25 in

Table 2.1 Age, sex and number of teeth present

Age-Group (years)	Number of individuals						No. teeth present
	M + F		M		F		
	No.	%	No.	%	No.	%	
16 - 19	155	(19.4)	69	(14.2)	86	(27.3)	25
20 - 24	195	(24.4)	115	(23.7)	80	(25.4)	25
25 - 29	117	(14.6)	74	(15.3)	43	(13.7)	24
30 - 34	90	(11.3)	52	(10.7)	38	(12.1)	23
35 - 39	67	(8.4)	49	(10.1)	18	(5.7)	21
40 - 44	68	(8.5)	52	(10.7)	16	(5.1)	20
45 - 73	108	(13.5)	74	(15.3)	34	(10.8)	16
All ages	800	(100.0)	485	(100.0)	315	(100.0)	23

16——19-year-olds to 16 in the 45——73-year age-group. Figure 2.3 shows the same data regrouped to allow comparison with similar findings reported by Todd & Whitworth (1974) from a representative sample of Scottish adults, selected for the 1972 survey of Adult Dental Health in Scotland (the ADHS survey—*see* Section 2.4).

Figure 2.4 shows the proportion of individuals in each age-group with ≥ 28 teeth present and the proportion with ≤ 12 teeth present. The 20——24-year age-group had the highest proportion of individuals (about one third) with ≥ 28 teeth and the numbers of teeth fell with increasing age until fewer than 5% of individuals in each age-group from 35——39-years onwards had ≥ 28 teeth. The proportion of the population with ≤ 12 teeth increased steadily with age, reaching 60.2% in the 45——73-year age-group.

2.3.2 Age and social class distribution

Figure 2.5 shows the social class distribution of the present study for both sexes together and, for comparison, similar data from the dentate participants in the ADHS survey. Individuals assigned to social class III comprised 43.9% of the sample with 13.7% in social classes I and II, and 39.4% in social classes IV and V. The remaining 3.1% could not be classified. The distribution of individuals within different social classes was broadly similar for all age-groups. Figures 2.6 and 2.7 show the social class distribution of males and females separately. When males are compared with females in the 'all ages' columns, their social class distributions are similar. However, when

Figure 2.3

Distribution of teeth present compared with the ADHS survey

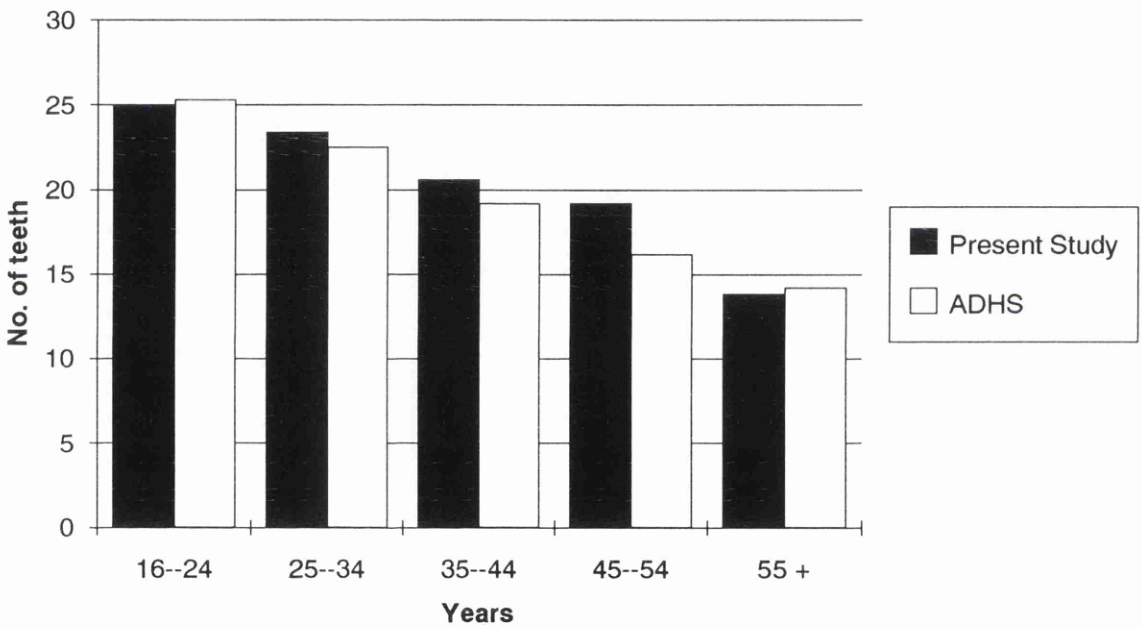


Figure 2.4

Number of teeth present by age

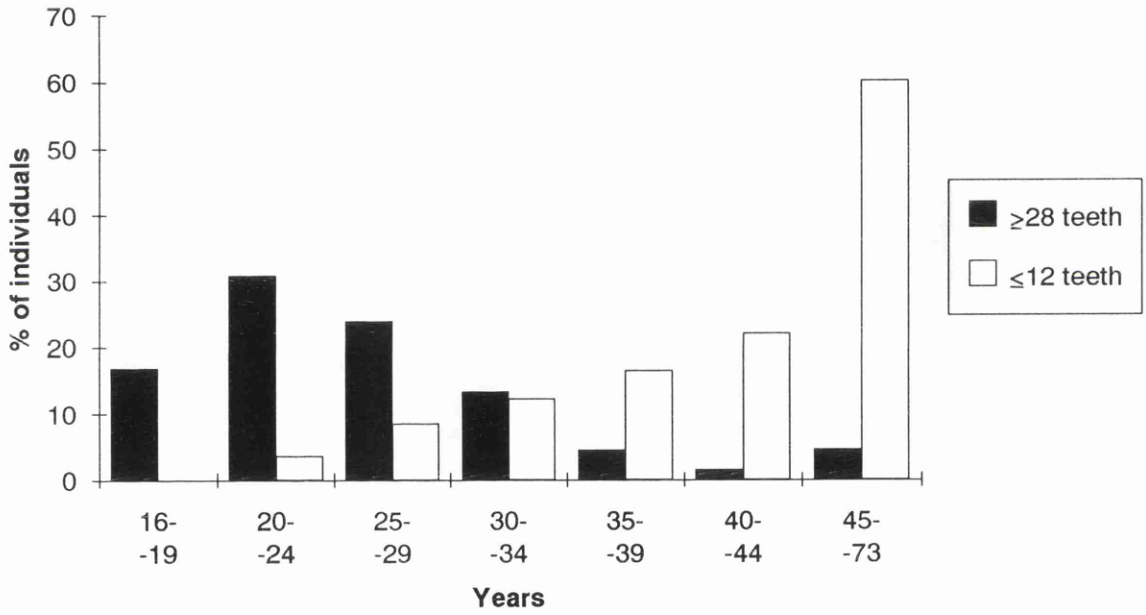


Figure 2.5

Social class distribution (both sexes)

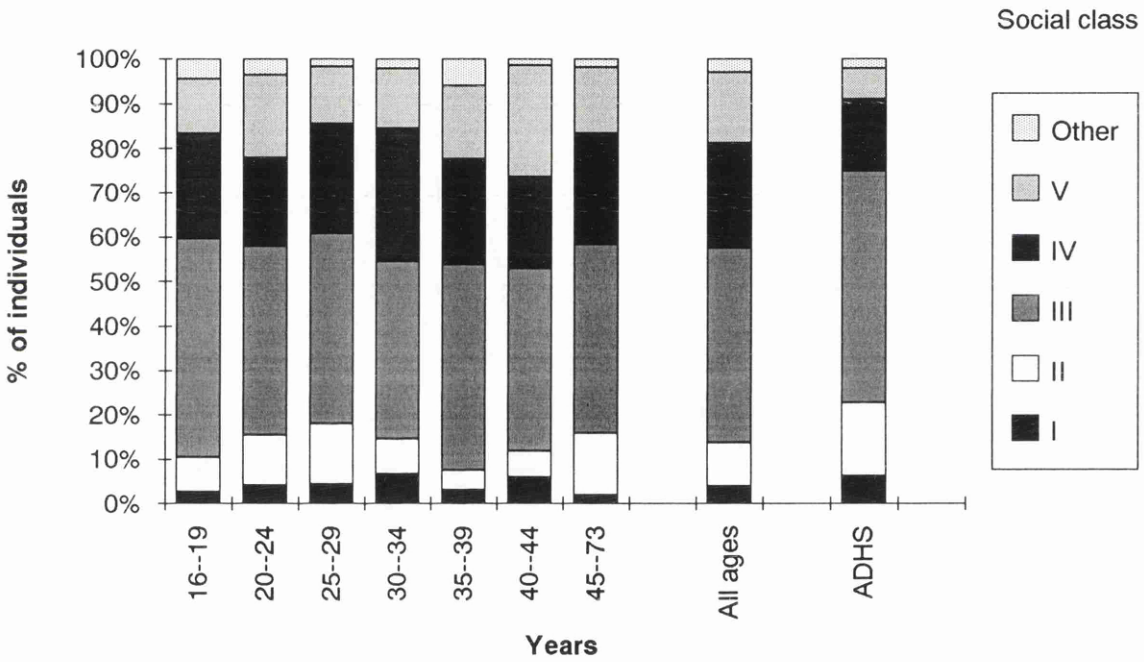


Figure 2.6

Social class distribution (males)

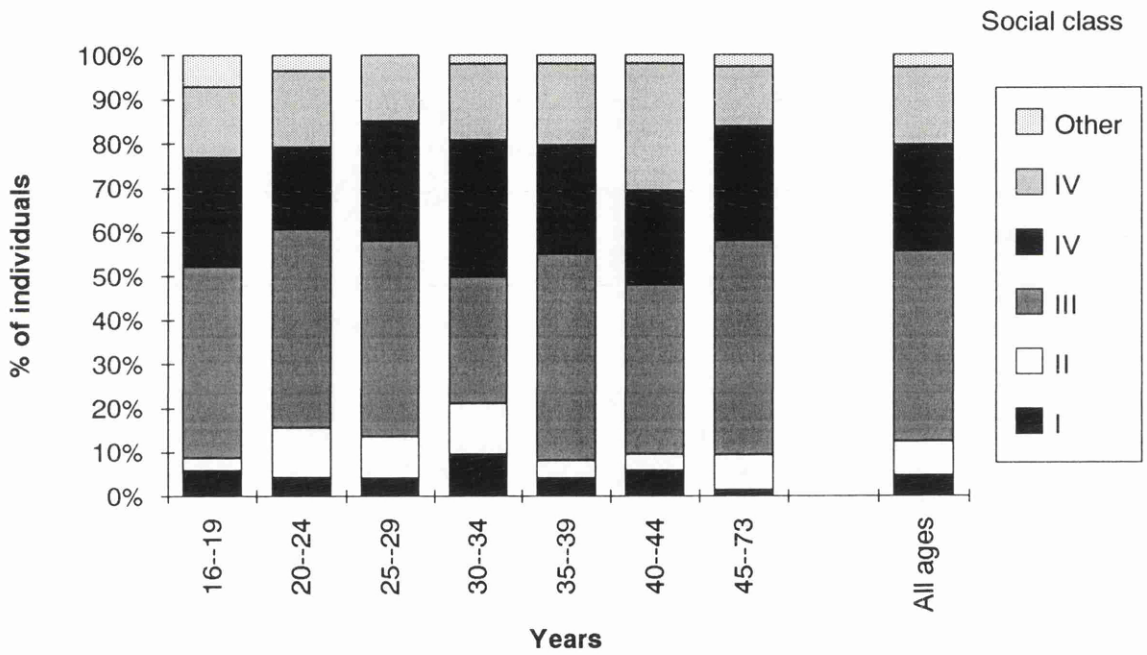
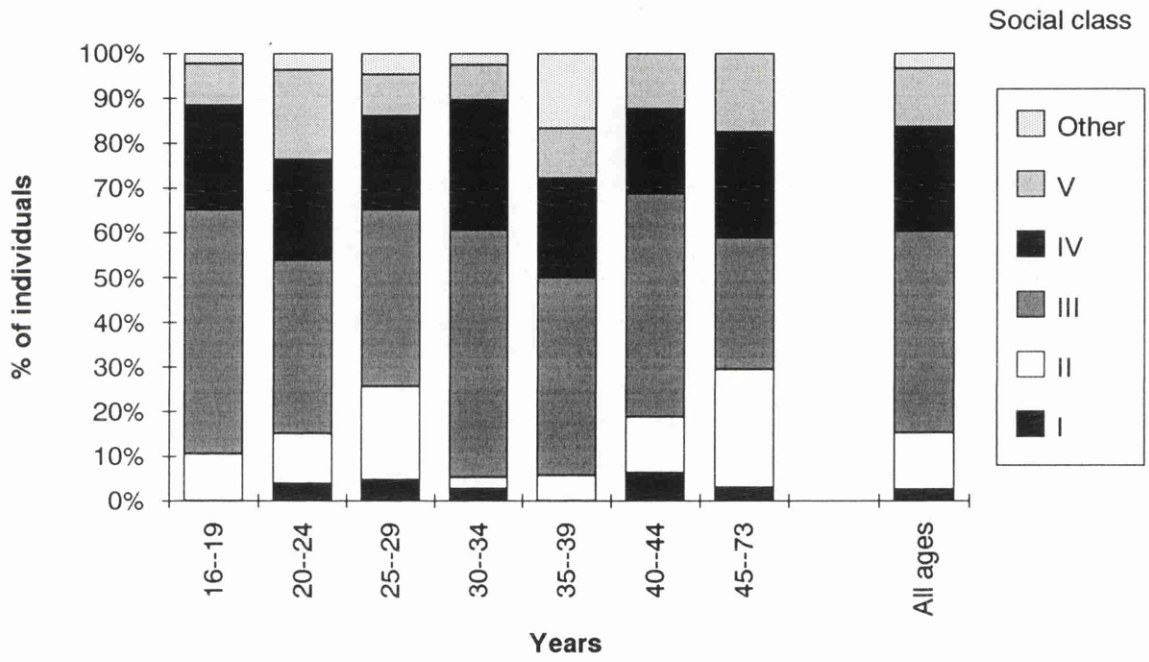


Figure 2.7

Social class distribution (females)



the social class distribution at different age levels is compared, either within or between sexes, a moderate amount of variation can be observed.

2.3.3 Reasons for attendance

Of the sample's treatment needs, 77.8% were met by referral to the Oral Surgery Department for extractions or minor oral surgery. A further 12.6% required restorative or endodontic treatment, while 6.4% either refused treatment, or were advised that none was necessary. Only 1.2% required periodontal treatment to meet their immediate needs.

2.3.4 Measurability

Owing to overlapping and poor contrast in certain regions of the dentition, it was not possible to identify the bone margin at all proximal tooth surfaces. Thus, only 78.6% of proximal surfaces were measurable. However, 89.8% of teeth had at least one proximal surface where the bone margin could be identified and all these teeth were given a bone loss score.

There were wide variations in the frequency with which the mesial and distal surfaces of different tooth types could be measured (Figures 2.8 and 2.9). The maxillary canine and premolar surfaces were least often measurable. For example, the bone margin on the mesial aspect of the maxillary first premolar could be identified for only 13.5% of these teeth. The distal bone margin was identified for 41.2% of maxillary first premolars, and only 44.6% of these teeth could be given a bone loss score, based on at least one measurable surface.

Figure 2.8

Percentage measurability of maxillary surfaces

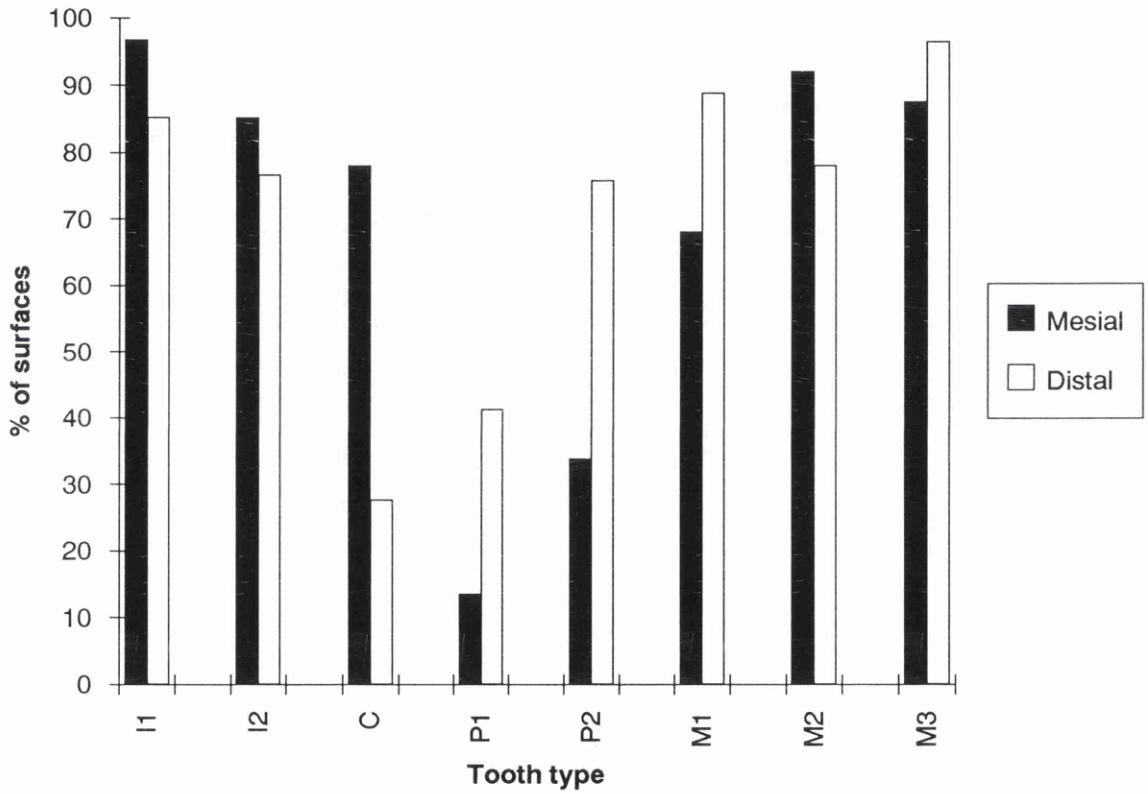
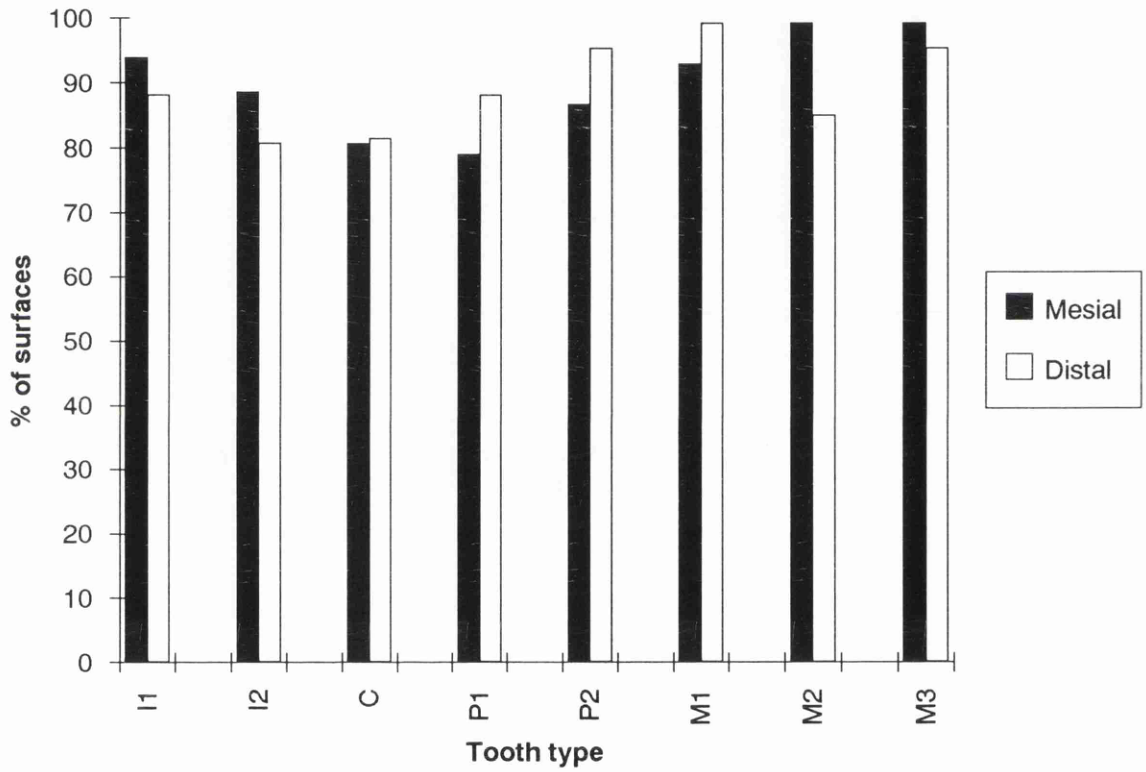


Figure 2.9

Percentage measurability of mandibular surfaces



For all teeth, other than upper canines and premolars, bone loss scores could be obtained for at least 90% of each tooth type.

2.3.5 Reproducibility

Among the 80-patient subsample, 83.7% of tooth surfaces were awarded the same score on the two occasions they were measured, and 99.6% of surface scores did not differ by more than one grade when scored a second time.

2.3.6 Analyses based on mean bone scores

Marginal bone loss generally increased with increasing age in both sexes, and was more advanced in males than in females at all age levels except in the 16—19-year age-group (Figure 2.10). After log transformation of the data, regression analysis revealed a highly significant association between age and bone loss (r^2 = 47.3%, $p < 0.001$) and a modest but statistically significant association between sex and bone loss (r^2 = 2.9%, $p < 0.001$).

Figure 2.11 depicts mean bone scores grouped according to sex and social class. Among males, the amount of bone loss was related to social class: those in social classes IV and V having more bone loss than those in social class III who, in turn, had more bone loss than those in social classes I and II. No such relationship was apparent among females where there was slightly less variation in bone scores between the social class groups, without any pattern apparent. Within-sex comparison of mean bone scores revealed significant differences only

Figure 2.10

Distribution of mean bone loss scores by sex

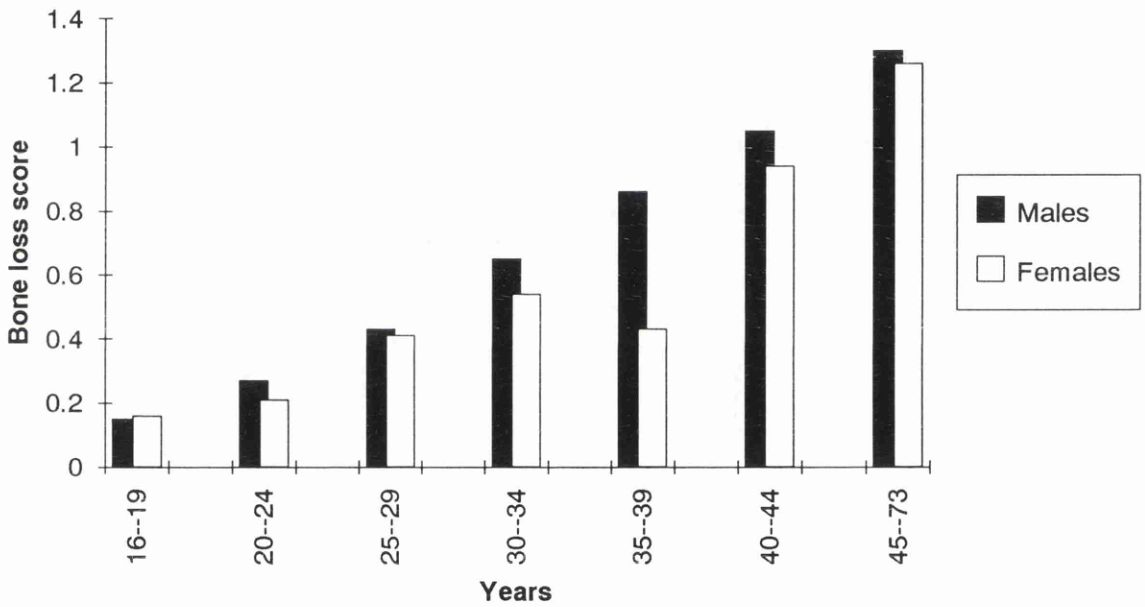
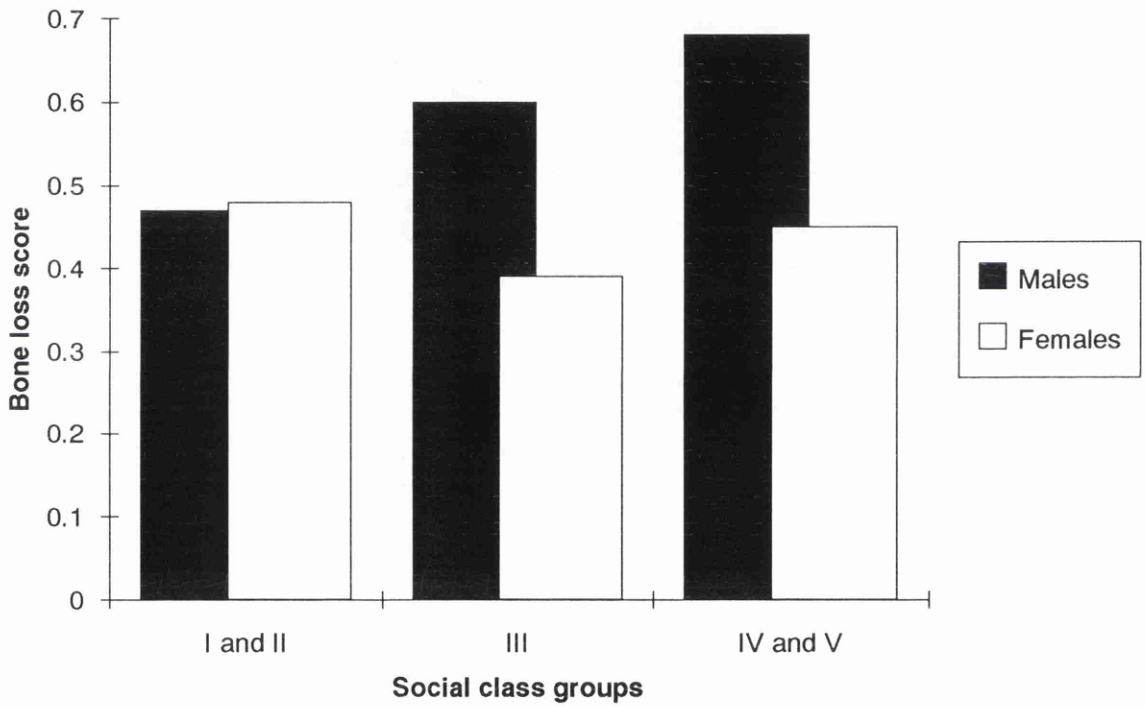


Figure 2.11

Distribution of mean bone loss scores by sex and social class



between social class I/II males and social class IV/V males (t = 2.93, $p < 0.01$).

Table 2.2 shows the proportion of individuals with no bone loss (mean bone scores = '0'), bone loss averaging less than 25% of optimum height (mean bone scores between '0' and '1'), and bone loss averaging 25—50% of optimum height (mean bone scores between '1' and '2'). No person exhibited bone loss averaging more than 50% of optimum height (mean bone score greater than '2'). The great majority, at all age levels, had mean bone scores of less than '1', while a small, but with age, increasing minority had bone scores between '1' and '2'.

The findings of the present study are compared, in Figure 2.12, with those of Björn (1971) by illustrating the progression of *distal* bone loss with age (It is unclear why Björn chose to present only distal bone score data). The 410 males of the present investigation, aged 20—64 years, are compared with 1,042 male 'white collar' workers, aged 20—64 years, and 535 male 'blue collar' workers, aged 20—65 years, employed by a large shipbuilding yard in Malmö, Sweden.

2.3.7 Analyses based on numbers of teeth affected at different levels of severity

Table 2.3 and Figure 2.13 illustrate the prevalence of bone loss beyond different bone loss thresholds for different age-groups (i.e. the proportion of the population affected by increasing degrees of severity of periodontal disease). Thus, while 84.5% of 16—19 year-olds were affected by bone loss of some degree (i.e. bone loss ≥ Grade 1), 14.2% were affected by

Table 2.2 Distribution of individuals according to mean bone scores

Age-group (years)	Number of Individuals					
	Mean bone score = 0		Mean bone score = 0 - 1		Mean bone score = 1 - 2	
	No.	(%)	No.	(%)	No.	(%)
16 - 19	24	(15.5)	131	(84.5)	0	(0)
20 - 24	8	(4.1)	187	(95.9)	0	(0)
25 - 29	1	(0.9)	115	(98.3)	1	(0.9)
30 - 34	1	(1.1)	88	(97.8)	1	(1.1)
35 - 39	1	(1.5)	64	(95.5)	2	(3.0)
40 - 44	0	(0)	64	(94.1)	4	(5.9)
45 - 73	0	(0)	96	(88.9)	12	(11.1)
All ages	35	(4.4)	745	(93.1)	20	(2.5)

Figure 2.12

Age development of mean distal bone loss scores in Sweden and Scotland

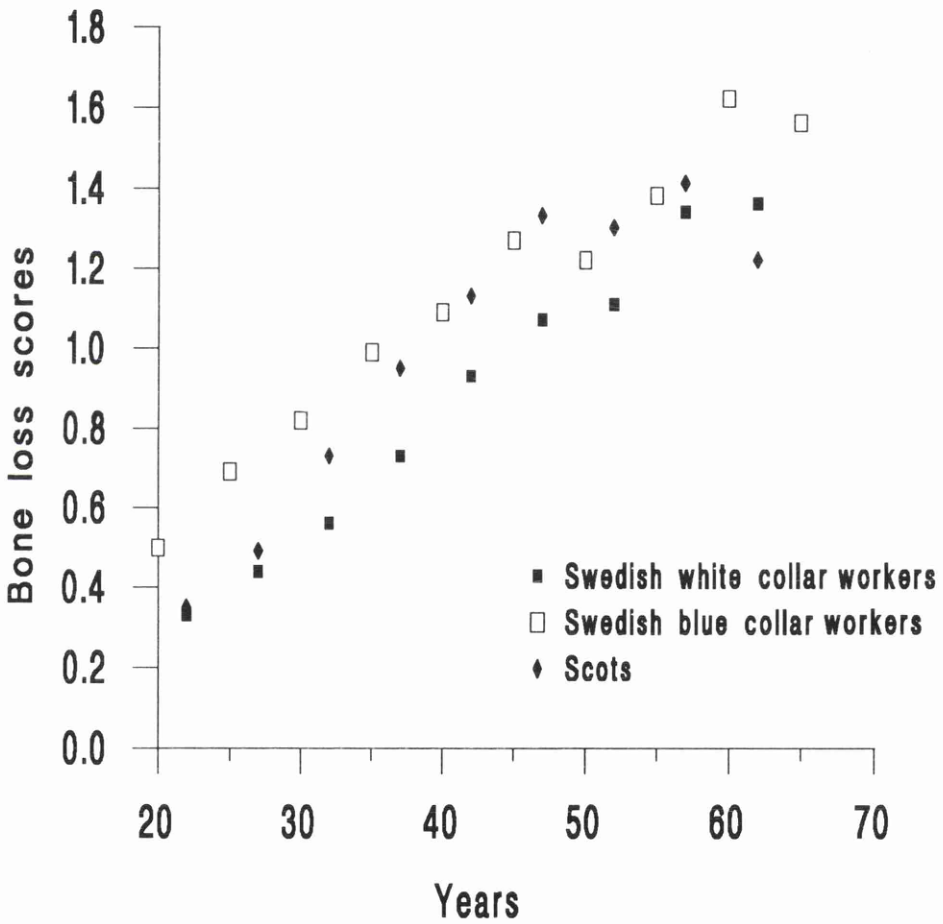
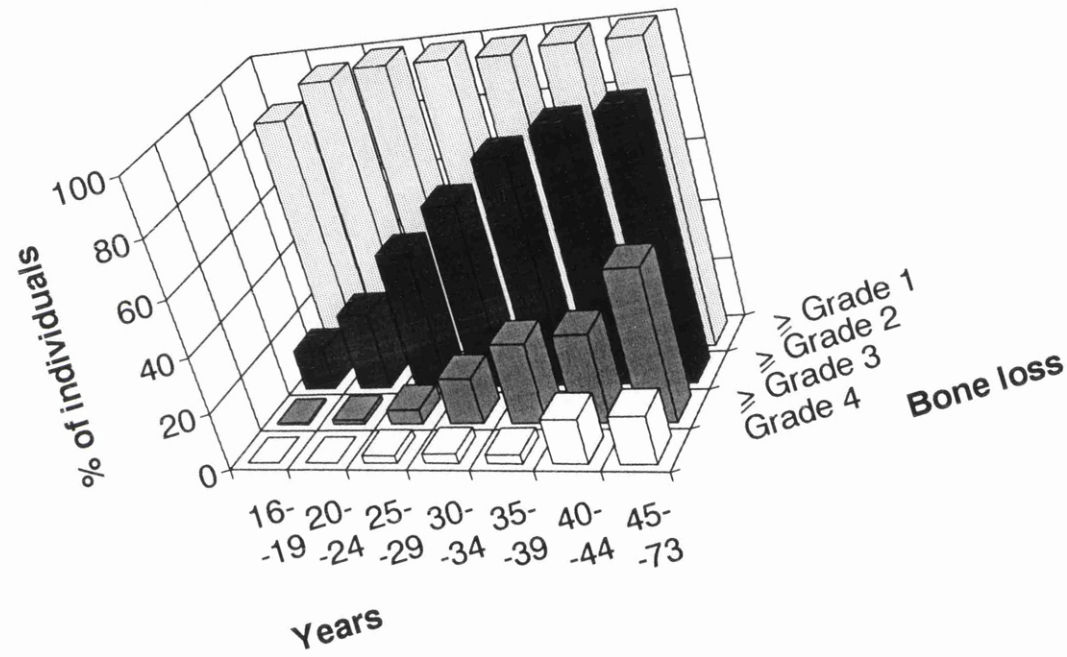


Table 2.3 Prevalence of bone loss beyond different bone loss thresholds

Age-group (years)	Percentage of Individuals			
	Bone loss at one or more surfaces			
	≥ Grade 1	≥ Grade 2	≥ Grade 3	= Grade 4
16 - 19	84.5	14.2	0.6	0
20 - 24	95.9	26.6	1.5	0
25 - 29	99.1	47.0	4.3	2.6
30 - 34	98.9	62.2	15.6	3.3
35 - 39	98.5	76.1	26.9	3.0
40 - 44	100.0	83.8	29.4	14.7
45 - 73	100.0	87.0	50.0	15.7
All ages	95.6	49.1	14.4	4.4

Figure 2.13

Prevalence of bone loss



bone loss ≥ Grade 2, 0.6% by bone loss ≥ Grade 3 and none by bone loss = Grade 4. At the opposite end of the age spectrum, all 45—73 year-olds were affected by some degree of bone loss; 87% by bone loss ≥ Grade 2, 50% by bone loss ≥ Grade 3 and 15.7% by bone loss = Grade 4.

The numbers of teeth with bone loss in different age-groups are illustrated in Figure 2.14. This shows that, up to the age of 35—39 years, the number of teeth affected increased with increasing age, with 26—36% of the population in each age-group having 6—10 affected teeth and almost two thirds of individuals in the three highest age-groups having 11 or more affected teeth.

The proportion of teeth affected by different degrees of bone loss increased with increasing age (Table 2.4 and Figure 2.15). In the 30—34-year age-group, almost half of the dentition was affected by some bone loss; 37.1% by Grade 1 bone loss, 11.2% by Grade 2 bone loss and 1.5% by Grades 3 or 4 bone loss. The proportion of teeth affected by bone loss scores of '3' and '4' was very low in all age groups, reaching a maximum of 8.4% per person, on average, in the 45—73-year age-group.

2.4 Discussion

It is well established that periodontal disease increases with age, being more advanced in males and in lower social class groups (Waerhaug, 1966). Careful consideration must, therefore, be given to the demographic and social

Figure 2.14

Distribution of individuals by number of teeth with bone loss

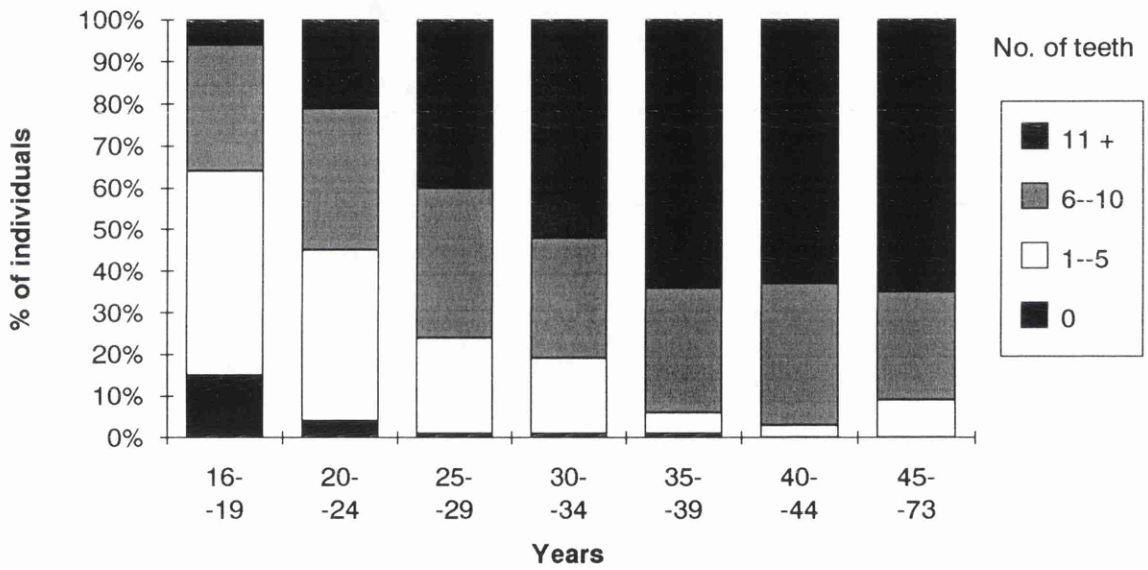
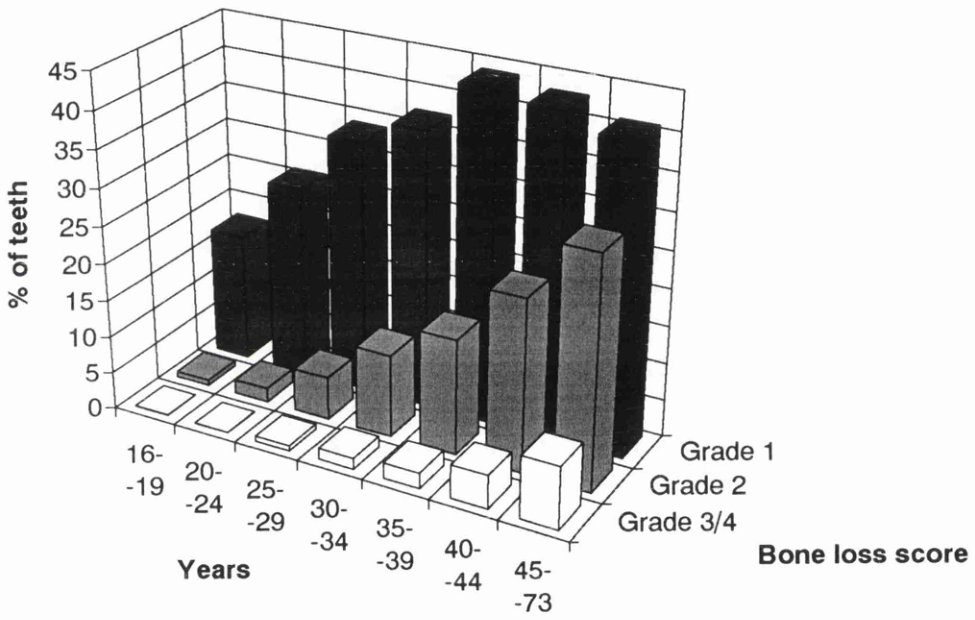


Table 2.4 Distribution of teeth with various degrees of bone loss by age

Age-group (years)	Percentage of Teeth			
	Bone Loss			
	Grade 1	Grade 2	Grades 3/4	All Grades
16 - 19	17.4	0.8	0.07	18.3
20 - 24	25.8	1.9	0.06	27.8
25 - 29	33.9	5.8	0.7	40.4
30 - 34	37.1	11.2	1.5	49.8
35 - 39	43.9	15.6	2.1	61.6
40 - 44	42.9	23.2	4.5	70.6
45 - 73	40.9	31.0	8.4	80.3
All ages	30.7	8.8	1.6	41.0

Figure 2.15

Distribution of teeth with various degrees of bone loss by age



characteristics of the population being investigated.

The choice of study population was influenced by the report of Stephen, McLundie & Kennedy (1971), describing the sociological aspects of attendance at Glasgow Dental Hospital and School, based on a survey of 1000 unselected casual patients. Thus, it appeared that this population of casual attenders might yield a sample, appropriately distributed by age, sex and social class, to ascertain the periodontal disease characteristics of the general population.

The age distribution of the present study population was, as anticipated, heavily weighted towards young people; the larger numbers in the younger age groups, among whom less disease was expected, being necessary to establish accurate prevalence data.

In attempting to confirm that adequate representation of the age-specific population subsets had been achieved, certain characteristics of the sample were compared to those of the multi-stage probability sample of Scottish adults, selected to take part in the 1972 survey of adult dental health in Scotland (ADHS), the findings of which were reported by Todd & Whitworth (1974). That survey was carried out by the Social Survey Division of the Office of Population Censuses in collaboration with the Scottish dental schools. A total of 3074 individuals were selected by a random sampling procedure: 2463 were subsequently examined. This figure was downweighted to 2076 to compensate for an over-representation from the Highlands and Islands region. The 1170 subjects with teeth (downweighted figure) in the ADHS survey may, therefore, be considered as a

probability sample of the population of dentate Scottish adults, 16 years-of-age and over. Periodontal data from the ADHS survey are reviewed in Chapter 1.

With regard to the sex ratio of the present study population, 60.6% were male, compared to 50.5% of the dentate adults in the ADHS survey. The male : female ratio, furthermore, varied considerably from the youngest age group, where females predominated, to the 40—44-year age-group where males outnumbered females by 52 : 16 (Table 1). Since a small sex difference in the occurrence of periodontal disease has been reported previously (Waerhaug, 1966), the present data, which combine males and females, must be viewed in the light of these unequal and variable sex ratios.

Compared to data from the ADHS survey of dentate individuals, the social class distribution of the present study was weighted slightly more towards the lower social classes (Figure 2.5). In addition, the social class distributions depicted in Figures 2.6 and 2.7 showed some variations between age-groups when males (Figure 2.6) or females (Figure 2.7) were considered separately, perhaps due to the relatively small numbers present in some age-groups. These differences were reduced considerably when male and female data were aggregated (Figure 2.5). Furthermore, when males of all ages (Figure 2.6) were compared to females of all ages (Figure 2.7), the social class distributions were very similar. Thus, the effect of social class variation on the prevalence and severity of bone loss at different age levels for males and females combined, should be negligible.

The present study population was formed of individuals who were actively seeking dental attention, many of them for relief of pain, suggesting that higher than 'normal' levels of dental and periodontal disease, and more evidence of neglect might be found. If this were the case, the study population should have more missing teeth than a representative sample of the general population. However, Figure 2.3 shows clearly that there were close similarities in the number of teeth present between this study's subjects and the dentate individuals of the ADHS survey. Indeed, in three out of five age-groups, the average number of teeth present was slightly greater in the present study.

Since most dental care in Scotland, as elsewhere, is provided in general practices and not in hospitals, the composition of the sample with regard to dental attendance habits cannot be regarded as typical of the general population. Thus, the study population may have been dominated by sporadic dental attenders. However, it has been reported that, in 1978, as many as 62% of Scottish dentate adults did not attend a dentist regularly (Todd *et al*, 1982). Besides, a study of a large Swedish population has revealed that periodontal conditions in patients receiving regular dental care were similar to those who attended sporadically (Björn, 1974).

It seems possible, when considering the likely effect of the small sex imbalance and slight weighting towards the lower social class groups, that the periodontal conditions of the present study participants should have been slightly worse overall than in the ADHS survey, and, therefore, in the general

population. Similarities in number of missing teeth, however, suggest that the participants' experience of *advanced* periodontal disease was similar to that of the general population.

A bone loss score could be obtained for 78.6% of proximal surfaces. This corresponds closely to the 81% measurability rate in the methodological study of Björn & Holmberg (1966). Indeed, the distribution of measurable surfaces is similar in both studies. A major cause of non-measurability was overlapping of upper canine and premolar teeth due to unfavourable projection angles in those segments of the arch. On the other hand, overlapping generally affected only the coronal half of the root so that, if substantial marginal bone loss existed, the bone margin could still be seen clearly in a zone where overlapping was absent. It is probable, therefore, that many of the teeth which could not be scored, due to radiographic overlap, were unaffected by severe bone loss, since their bone margins coincided with the overlapped area in the region of the cemento-enamel junction. Thus, it is unlikely that failure to identify the bone margin at every proximal surface could have resulted in serious under-recording of disease.

The findings that periodontal destruction generally increased in severity with increasing age, and that males, generally, were more severely affected than females, are in agreement with the ADHS survey report, with data reported by Sheiham (1969) for employed persons in London and Warrington, and with data from other industrial nations, as reviewed by Waerhaug (1966).

Sheiham (1969), however, found that Periodontal Index scores were greater in females than in males in both the 16—19- and 25—29-year age ranges, and some studies of marginal bone loss have observed higher scores in females than males up to the age of 30 years (Miller & Seidler, 1940; Helminen-Pakkala, 1968). In the present study, bone loss was greater in males than in females at all ages except at 16—19 years when, in common with the above reports, the sex difference was reversed. Curiously, both in the present study and that of Sheiham (1969), there was an apparent improvement in disease level in 35—39-year-old females, giving rise in both studies to the only substantial difference in disease severity between males and females across the whole age spectrum. This apparent anomaly was not commented upon by Sheiham: in the present study, the small number of females in the 35—39-year age-group may not have been representative of their age range in the population which was sampled.

To put in perspective the influence of gender, and allowing for the anomalous data for 35—39-year-old females, it should be noted that increments of bone loss in the present study were much greater between age levels, than between males and females of the same age range (Figure 2.10). Although there was a preponderance of male subjects, especially in the older age-groups, further detailed analyses of findings by sex were not carried out because large differences in periodontal status were not observed between the sexes.

In general, the relationship between bone loss and social class, observed in males, corroborates the findings of the ADHS survey, the findings of the study of British employed persons

(Sheiham, 1969) and data from other developed countries, as reviewed by Waerhaug (1966). The absence of any relationship between bone loss in females and their social class grouping probably reflects a weakness in the Registrar General's social classification, previously noted by Sheiham (1969), whereby the daughter or wife of a social class I male may be classified as social class III if she is employed as a clerical worker.

Although the amount of bone loss increased with age, there were, even so, only 20 individuals with *mean* bone loss scores equal to or exceeding 25% of optimum height, and these were all in the 25—50% range (Table 2.2). Of these individuals, 12 were in the 45—73-year age-group, amounting to 11.1% of that group. This shows that severe bone loss, whether affecting a few teeth in one mouth to a very severe degree, or most of the dentition to a moderately severe degree, was uncommon in the study population. These figures are of similar magnitude to those reported by Hugoson & Jordan (1982) who found that only 4.5% of individuals, 50—70 years-old, exhibited *generalised* bone loss between one-third and two-thirds of root length.

Comparison of periodontal conditions between different populations is fraught with difficulty unless the same diagnostic criteria are employed. Moreover, unless an objective diagnostic technique is used, any observed variations between populations may be attributable to inter-examiner variability. This might explain the substantially lower Periodontal Index scores in Sweden (Björn, 1974) compared to those in the United Kingdom (Sheiham, 1969). The present study, however, permits a more valid comparison of periodontal conditions in these countries since, in both studies to be

compared, the measurement of marginal bone loss was performed by the same technique of quantitative assessment. Thus, Figure 2.12 reveals that, in a socially mixed population of Scottish males, marginal bone loss developed with increasing age, in parallel with, and at a level of severity intermediate between, 'white' and 'blue collar' workers in Sweden (Björn, 1971). It would be reasonable to conclude, therefore, that the periodontal disease experience of these two populations was very similar.

Age-specific distributions of different degrees of bone loss (Table 2.3) show that the proportion of individuals with some bone loss (bone loss ≥ Grade 1), involving at least one tooth surface, affected 84.5% of the 16—19-year age-group, and increased with age to affect 100% of subjects in the oldest age-groups. Of course, it could be argued, either that the specificity of the measurement technique was too low to avoid a high proportion of false positive results, or that Grade 1 bone loss is of little clinical significance, especially when it was present almost universally in all age-groups. When the prevalence of bone loss ≥ Grade 2, ≥ Grade 3 and = Grade 4 are considered, it is apparent that the distributions become increasingly skewed with increase in disease level (Figure 2.13). Nevertheless, even the prevalence of bone loss ≥ Grade 2 was high, even among younger subjects. Almost half of the 25—29-year-olds and more than three-quarters of the 35—39-year-olds were affected. Furthermore, a high prevalence of bone loss ≥ Grade 3 was noted in the older age-groups, and 50% of 45—73-year-olds were affected. The finding that the age-specific distributions became increasingly skewed with increase

in disease level suggests that considerable variation in periodontal disease progress exists, so that only a small minority of individuals progress to Grade 4 bone loss. An alternative explanation, and one which is hard to disprove, would be that some teeth with progressive disease were removed from the calculation by extraction as bone loss advanced. However, on average, only five teeth were lost between 16—19 and 40—44 years. Distortion of the data by tooth loss before the age of 45, therefore, cannot account fully for the observed distributions which show a low prevalence of Grade 4 bone loss.

Just as the severity of bone loss increased with increasing age, the number of teeth affected by bone loss also increased (Figure 2.14). For example, the proportion of individuals in each age group with 11 or more affected teeth rose from 6% at ages 16—19 years to 63—65% in the three oldest age-groups. From the age of 35—39 years onwards, the levelling off in the number of affected teeth, and the small rise in the proportion of individuals with only 1—5 affected teeth most likely reflects the reduction in size of the dentition in these older individuals.

Although the proportion of teeth affected by bone loss was considerable at all ages (Table 2.4), and increased with increasing age from 18.3% of teeth at risk in 16—19-year-olds to 80.3% of teeth in 45—73-year-olds, most affected teeth exhibited Grade 1 bone loss. The distributions of teeth with Grade 2 bone loss and Grades 3—4 bone loss were highly skewed towards the higher age-groups (Figure 2.15): in 45—73-year-olds, 8.4% of teeth were affected by Grades 3—4 bone loss (i.e. at least 50% of optimum height on at least one surface),

but this is equivalent to only 1.2 teeth per person, on average.

Finally, it is necessary to acknowledge some well recognised drawbacks of cross-sectional epidemiological study. Firstly, interpretation of age-specific cross-sectional data, as if it were longitudinal, must be undertaken with great caution: older subjects in this survey grew up under very different conditions from those experienced by the younger ones. Age cohorts pass through the population slowly and the effects of previous preventive health behaviour and treatment patterns take a long time to work themselves out. The age development of marginal bone loss may, therefore, be quite different from what is portrayed in cross-sectional studies. Secondly, continuity of disease progression cannot be confirmed from cross-sectional data, and those teeth or individuals with moderate disease at an early age may not necessarily become part of the severe group at a later age. Finally, in common with other cross-sectional studies in industrialised countries, the prevalence and severity of periodontal disease in older individuals is likely to be an underestimate of their periodontal disease *experience*: since the reasons for tooth extraction are unknown, the degree of underestimation is an undetermined function of the number of teeth lost.

2.5 Conclusions

1. Although logistic and cost considerations dictated that the study population should comprise dental clinic out-

patients, there is evidence to suggest that periodontal conditions among these individuals would be only slightly worse than in the general population.

2. The measurement technique successfully recorded bone levels at 78.6% of tooth surfaces, corresponding closely with data from the originators' methodological study.
3. Marginal bone loss increased with age, and was, on the whole, slightly more severe in males and amongst males from the lower social class groups, in accordance with established beliefs. However, only a tiny minority of individuals had *generalised* bone loss averaging 25—50% of their optimum bone heights. This finding is explored further in Chapter 3.
4. Analysis of frequency distributions revealed that the development of bone loss with age was attributable both to an increase in affected surfaces and to progressive destruction at some of these surfaces.
5. Even if Grade 1 bone loss is rejected as clinically insignificant or is attributable to examiner error in a large proportion of cases, a high prevalence of marginal bone loss would still be present, based on a bone loss threshold value of Grade 2 (bone loss > 25% of optimum bone height). For example, almost half of the 25—29-year-olds and more than three quarters of the 35—39-year-olds were so affected, and the proportion increased with increasing age.
6. Bone loss, based on a threshold value of Grade 3, affected more than one quarter of the 35—39-year age-group, and again the proportion increased with increasing

age.

7. Grade 4 bone loss was uncommon except in the two oldest age-groups where 14.7—15.7% were affected.
8. Only a tiny proportion of *teeth* were affected by Grades 3 and 4 bone loss.
9. According to the present data, and ignoring the cohort effect, an individual living to be 45—73 years-old, had a 50% chance of at least one tooth with Grade 3 or 4 bone loss, and an 11% chance of *generalised* bone loss averaging 25—50% of optimum bone height.
9. In this population, before the age of 45 years, there was little evidence to support the view that marginal bone loss could be a significant cause of tooth loss.
10. Use of the 'Björn ruler' in the present study enabled the findings for males aged 20—64 years to be viewed alongside those of a comparable population of Swedish males. These different populations had similar marginal bone levels.

CHAPTER 3

THE 'HIGH RISK' GROUP IN PERIODONTITIS

3.1 Introduction

It is well established that dental bacterial plaque is the primary aetiological agent of inflammatory periodontal diseases. It is equally well accepted that some individuals have a much greater risk of suffering severe and widespread periodontitis (inflammatory destruction of supporting tissues) than others. There is, however, less agreement as to the size of the 'high risk' group, a factor of considerable importance in health care planning. If the high risk group is very small, say 10% or less of the population, then some means of identifying susceptible individuals while still at an early stage of the disease may allow more efficient use of limited treatment resources.

Ideally, to calculate the proportion of teeth and individuals experiencing advanced, destructive periodontitis, a longitudinal study is required in which the subjects are followed from puberty to old age. This is quite impracticable, of course, and conclusions must be drawn instead from cross-sectional studies or from the limited longitudinal work which is presently being undertaken.

The epidemiological data presently available to support the high risk concept of periodontitis come from several

sources: measurements of attachment loss and pocket depth, radiographic measurements of marginal bone loss and studies on extracted teeth.

Löe *et al.* (1986) carried out a 15-year longitudinal study of Sri Lankan tea labourers, initially aged 14—31 years, who were not accustomed to carrying out oral hygiene procedures. Periodontal destruction was widespread, due perhaps to racial factors or undernourishment, as well as high plaque levels. Yet, it was still possible to identify a 'high risk' group of 8% in whom periodontal disease resulted in loss of virtually the entire dentition by 45 years-of-age.

Baelum, Fejerskov & Karring (1986) reported cross-sectional findings in a population of adult Tanzanians, aged 30—69 years, who, like the Sri-Lankan labourers (above), harboured abundant quantities of dental plaque. In contrast to the Sri-Lankans, only 10% of tooth surfaces among the Tanzanians had advanced loss of attachment. Furthermore, relatively few individuals accounted for most of the advanced loss of attachment. Similar findings were reported in a more recent study of adult Kenyans (Baelum, Fejerskov & Manji, 1988).

Another way of assessing the contribution of periodontal disease to tooth loss is to examine the periodontal condition of teeth in patients attending for full clearance. This has been done by dental panoramic tomograph assessment (Linden, 1988) and by direct measurement of loss of attachment on extracted teeth (Bouma, Schaub & Van de Peol, 1985). Thus, Linden (1988) showed that, among 373 individuals, at the time

of full clearance, only 15% had more than 50% of their teeth with more than 50% reduction in bone height; 38% of patients 40 years-and-over had no teeth with this amount of bone loss. Similar findings in a Dutch community are reported by Bouma *et al.* (1985).

All of the studies cited above point to the existence in most societies of a small group of individuals at high risk of losing a large number of teeth through periodontitis. Indeed, drawing on his own research findings and on other surveys in which pocket depths were used to describe disease severity, Schaub (1984) concluded '..... that 10—15% of adult populations with natural teeth will lose teeth because of progressive periodontal disease and that approximately half of them will lose most of their teeth'.

Jenkins & Mason (1984) reported the findings of a dental panoramic tomograph study of marginal bone loss in 800 casual attenders at Glasgow Dental Hospital and School (*see* Chapter 2). It was observed that only 1.6% of all teeth in individuals of all ages exhibited bone destruction exceeding 50% of optimum bone height (Table 2.4). In the present report, the results of that study are further analysed to describe the distribution of teeth in that category within the 800-subject population, as a contribution to the debate on the high risk concept of periodontitis.

3.2 Material and methods

Details of the sample, research methods and examiner variability have been described in Chapter 2. The data presented in this chapter relate to the prevalence within the 800-subject sample of bone loss exceeding 50% of optimum bone height, referred to subsequently as advanced bone loss (ABL). The subsample, affected by ABL, comprised 115 individuals.

3.3 Results

3.3.1 Measurability

Due to overlapping and poor contrast in certain regions of the dentition, 21.4% of proximal surfaces in the 800-subject sample were unmeasurable. In the subsample of 115 individuals, the proportion of unmeasurable surfaces was only 15.7% However, all but 10.2% of teeth in the 800-subject sample and all but 5.9% of teeth in the 115-subject subsample had at least one proximal surface where a score could be given.

3.3.2 Linear regression analysis

The age of the individual and the percentage of his teeth affected by ABL showed a highly statistically significant correlation (correlation co-efficient = 0.36, $p < 0.0001$). There was a similarly highly significant negative correlation between the number of teeth present and the age of the individual (correlation co-efficient = - 0.55, $p < 0.00001$).

Also, the percentage of teeth affected by ABL and the number of teeth remaining in the individual revealed a highly significant negative correlation (correlation co-efficient = - 0.6, $p < 0.00001$).

3.3.3 Age by advanced bone loss

Table 3.1 shows the distribution by age-group of individuals: (a) within the 800-subject sample; (b) within the subsample affected by advanced bone loss (ABL); and (c) within a further subsample affected by generalised advanced bone loss (GABL), defined as at least 50% of remaining teeth with advanced bone loss. Within the 800-subject sample, 115 individuals (86 males, 29 females) were identified with ABL, of whom eight were affected by GABL. In percentage terms, 14.4% were affected by ABL and 1% by GABL. The prevalence of ABL varied from 0.6% in the 16—19-year age-group to 54.1% in the 50—73-year age-group, and GABL was absent until 40 years-of-age, when it occurred in 3.9% of 40—49-year-olds and 5.4% of 50—73-year-olds.

Table 3.2 shows the proportion of individuals in each age-group of the 800-subject sample accounting for 75% of the observed advanced bone loss in that group. Thus, with increasing age, an increasing proportion of individuals accounted for most of the advanced bone loss. However, even in the oldest age-group, 50—73 years, this amounted to a relatively small proportion of individuals.

Table 3.1 **Distribution of patients by age showing (a) the number affected by advanced bone loss (ABL), (b) the number affected by generalised advanced bone loss (GABL) and (c) the total number of patients with the average number of teeth present. Figures in parentheses refer to the percentage of all patients in that age group studied.**

	(a) Patients with ABL		(b) Patients with GABL		(c)	
Age group	No.	%	No.	%	Total patients No.	Teeth present No.
16 - 19	1	(0.6)	0	(0)	155	25
20 - 24	3	(1.5)	0	(0)	195	25
25 - 29	5	(4.3)	0	(0)	117	24
30 - 34	14	(15.6)	0	(0)	90	23
35 - 39	18	(26.9)	0	(0)	67	21
40 - 49	34	(33.3)	4	(3.9)	102	19
50 - 73	40	(54.1)	4	(5.4)	74	15
All ages	115	(14.4)	8	(1.0)	800	23

Table 3.2 The proportion of patients within the 800-patient sample accounting for 75% of the total number of teeth with advanced bone loss.

Age group (years)	Proportion
30 - 34	8%
35 - 39	15%
40 - 49	15%
50 - 73	28%

3.4 Discussion

Various techniques have been employed in OPG radiographic surveys to quantify marginal bone loss (Björn & Holmberg, 1966; Helminen-Pakkala, 1968; Jenkins & Mason, 1984; Kaimenyi & Ashley, 1988). The present study used the proportional principle relating the actual bone level to the optimum bone height as described by Björn & Holmberg (1966). Other investigators have measured the amount of bone loss in millimetres, using the cemento-enamel junction as a fixed reference point (Kaimenyi & Ashley, 1988). The respective advantages and limitations are discussed in Section 2.1.1 and elsewhere (Björn & Holmberg, 1966; Kaimenyi & Ashley, 1988). Although radiographs cannot be used in isolation to diagnose current disease, they can demonstrate the previous occurrence of periodontitis by its destructive effect on supporting bone.

A bone loss score could not be obtained for 21.4% of surfaces in the 800-subject sample, and 15.7% of surfaces in the 115-subject subsample. These failure rates are comparable to those of other dental panoramic tomograph surveys of periodontal bone loss (Björn & Holmberg, 1966; Kaimenyi & Ashley, 1988; Linden, 1988). A major cause of non-measurability was overlapping of upper canine and premolar teeth, due to unfavourable projection angles in those segments of the arch. Overlapping generally affected only the coronal half of the root, so that, if substantial marginal bone loss existed, the bone margin could still be clearly seen in an area where overlapping was absent. It is likely, therefore, that

many of the teeth which could not be scored due to radiographic overlap were unaffected by severe bone loss, since their bone margins coincided with the overlapped area in the region of the cemento-enamel junction. Thus, the observed prevalence of advanced bone loss in this population is unlikely to be seriously affected by the failure to score all of the proximal surfaces. The reduced frequency of non-measurability in the 115-subject subsample with advanced bone loss supports this assumption.

The present study, in agreement with others (Lavstedt & Eklund, 1975; Palmqvist & Sjödin, 1986; Papapanou, Wennström, & Gröndahl, 1988), shows a highly significant negative correlation between the percentage of teeth affected by advanced bone loss and the number of remaining teeth. This suggests that, despite underestimating the total number of teeth affected by advanced bone loss, due to absent teeth, it was still possible to identify the 'at risk' individuals by the condition of their remaining teeth.

Table 3.1 shows that advanced bone loss was highly prevalent in the older age-groups in this population, in common with many other published studies of attachment loss or periodontal bone destruction. For example, Okamoto *et al.* (1988) have reported prevalence data from a periodontal survey of a random sample of urban Japanese: the prevalence of attachment loss greater than 6 mm rose from 4% of 20—29-year-olds to 45% of 50—79-year-olds. Likewise, in a radiographic study of Swedish dental school patients, Papapanou *et al.* (1988) reported a high prevalence of advanced bone destruction

(a bone level value of 6 mm or more from the cemento-enamel junction), increasing with increasing age to 83% of 75-year-olds.

These data invite comparison with prevalence data of deep pockets, as reported in studies utilising the Community Periodontal Index of Treatment Needs (CPITN) (Pilot & Barmes, 1987). Thus, the prevalence of deep pockets in 35—44-year-olds in 12 European countries varied from 2% to 18% of individuals. This is lower than the prevalence rates for advanced bone and attachment loss described above, presumably because gingival recession often occurs as periodontitis progresses, so that pocket depths tend not to reflect the full extent of periodontal destruction (Baelum *et al.*, 1986).

While the prevalence of advanced bone loss was high in the present study, generalised advanced bone loss, that is affecting more than 50% of the remaining dentition, was uncommon. This affected only 3.9% of 40—49-year-olds and 5.4% of 50—73-year-olds. It must be remembered, however, that edentulous individuals were excluded from the present study, so that the low percentages recorded above may not accurately reflect the proportion of individuals in the general population who experience generalised advanced bone loss.

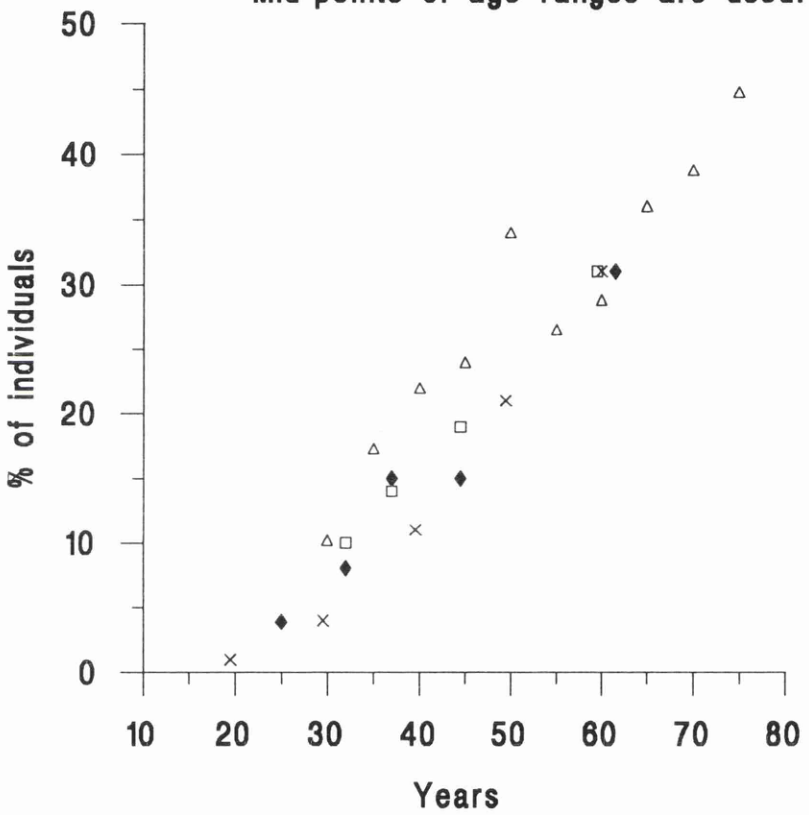
Another way of assessing how advanced bone loss is distributed within a population is to calculate the cumulative distribution of individuals according to the cumulative proportion of teeth with advanced bone loss. The data thus obtained in the present study (Table 3.2) are in close agreement with the findings of other studies which have

employed this method of analysis (Baelum *et al.*, 1986, 1988; Papapanou *et al.*, 1988). Figure 3.1 compares the results of the present survey with similar studies by Baelum *et al.* (1986, 1988) and Papapanou *et al.* (1988), showing the proportion of individuals accounting for 75% of the total number of surfaces or teeth affected by advanced destruction in different age-groups. Baelum *et al.* (1986, 1988) carried out clinical periodontal surveys of 170 adult Tanzanians and 1,131 adult Kenyans, respectively, and their findings with respect to surfaces affected by at least 7 mm loss of attachment are recorded in Figure 3.1. Papapanou *et al.* (1988) carried out a radiographic survey of bone levels in a population of 531 Swedish dental school patients, and the data presented in Figure 3.1 relate to proximal tooth surfaces with bone level values 6 mm or more from the cemento-enamel junction. Thus, Figure 3.1 demonstrates a remarkable degree of agreement between all four studies, showing that a small, but with age increasing, minority of individuals accounted for most of the advanced periodontal destruction. At about 60 years-of-age, approximately 30% of individuals accounted for 75% of the advanced periodontal destruction in that category.

In conclusion, the present study confirms the existence of a 'high risk' group in this population of unreferred Dental Hospital attenders and, by cautious extrapolation, in the Strathclyde Region of Scotland. However, the size of the 'high risk' group was somewhat larger than might have been anticipated from reports of epidemiological studies based on pocket depths. The present study, in common with others from

Figure 3.1

Combination of data from four different studies on percentage of individuals in each age group accounting for 75% of the total number of sites affected by advanced periodontal destruction. Mid-points of age ranges are used.



♦ Jenkins & Kinane (present study) > 50% bone loss
△ Papapanou et al (1988) ≥ 6mm bone level reduction
□ Baelum et al (1986) ≥ 7 mm attachment loss
× Baelum et al (1988) ≥ 7 mm attachment loss

various parts of the world, in which periodontal destruction was assessed by loss of attachment measurements or by radiographic assessment of marginal bone loss, demonstrated a high prevalence of advanced destruction, but also showed that relatively few individuals in each age-group account for most of the advanced periodontal disease.

The finding, that 28% of 50—73-year-olds accounted for 75% of the advanced destruction seen in that age-group, suggests that three-quarters of advanced periodontal disease could be prevented by targeting an effective preventive strategy on the 28% of individuals specially at risk. The question remains, whether an acceptable preventive strategy can be devised and whether it is possible to establish a simple method of identifying the 'at risk' group. At the present time there would appear to be no reasonable alternative but to advocate periodic thorough examination for all patients, treatment of all lesions while still at an early stage, and appropriate dental health education.

CHAPTER 4

A CROSS-SECTIONAL ASSESSMENT OF CARIES AND PERIODONTITIS RISK WITHIN THE SAME SUBJECT

4.1 Introduction

If the occurrence of dental caries and chronic periodontitis were related, it would be feasible to predict periodontitis by examining caries experience, since caries is a disease which occurs earlier in the lifetime of the individual.

Although the relationship between dental caries and chronic periodontitis has been addressed by many previous workers, their conclusions have been contradictory. In a study of Down's syndrome patients, an inverse association between periodontitis and smooth surface caries was found (Barnett *et al.*, 1986). In another selected patient group, juvenile periodontitis patients, fewer proximal caries lesions were detected than in the control group without periodontitis (Fine, Goldberg & Karol, 1984). While these two studies suggest a negative relationship between caries and periodontitis for these specific disease groups, their findings would not necessarily be applicable to the general population. A negative association between caries and periodontitis was also reported by Sewõn *et al.* (1988) who compared the caries level of periodontitis-free individuals with a periodontitis-affected group in a cross-sectional study. This study, unfortunately,

is flawed in that the authors selected and contrasted, on an *a priori* basis, two groups which they designated 'periodontitis-free' (n=291) and 'advanced periodontitis' (n=125), from an original population of 1,105 Finns. They excluded individuals with mild bone loss or with less than six teeth and those for whom no sex- and age-matched controls existed. It is not clear from the disparate numbers in the designated groups how they arrived at matched pairs, and this study tends to pose more questions than it answers.

In contrast to the above findings, Skier & Mandel (1980) examined the periodontal status of caries-resistant and caries-susceptible individuals in 40 sex- and age-matched pairs and found no significant differences between the two groups. However, they did find that female caries-resistant subjects had a lower level of periodontitis and stated that this difference may have arisen because of the small numbers examined. This study is, therefore, somewhat equivocal and, although adequately designed, suffers from a lack of subjects. Although Ainamo, Ankkuriniemi & Parviainen (1980) looked at a large population (3344 Finnish military conscripts) and found no relationship between caries and periodontal disease within the same individual, the mean age of subjects was only 20.3 years. Thus a high periodontal disease prevalence would not be expected.

In summary, the published research on this subject suffers from major shortcomings: none of the studies cited above has involved a sufficient number of subjects spanning a large age range which is representative of the general population.

Jenkins & Kinane (1989) (*see* Chapter 3) have recently reported the results of a radiographic study of 800 unreferred dental outpatients (aged 16-73 years) which determined a high risk group for periodontitis in several age categories of patients. The present report extends that study by comparing the susceptibility to periodontitis and caries within the same individual.

4.2 Material and methods

4.2.1 The study population

The study population comprised 800 unreferred dentate individuals (485 males, 315 females) of 16 years-and-over, presenting with a variety of dental complaints to be examined at Glasgow Dental Hospital and School during the period from February to May 1978 as 'casual' patients (*see* Section 2.2.1 for further details).

4.2.2 Methods

A rotational tomographic view was taken for each patient using the Siemens Orthoceph 3. The height of alveolar bone at each approximal surface was calculated with a transparent ruler using the crown tip and root apex as reference points. The ruler was calibrated to score bone loss in 'quarters' of optimum bone height, and a periodontitis risk score was derived by assessing bone loss in quarters of optimum bone height. All the bone height determinations were carried out by one examiner

(the author) (*see* Section 2.2 for further details). For this investigation, comparing caries and periodontitis risks, a mean value was obtained for each subject, based on all surfaces to which a bone loss score had been attributed. Similarly, the caries risk was determined radiographically (by another examiner) from the total of decayed or filled teeth (DFT) as a percentage of the total teeth measured. Thus:

$$\textbf{Periodontitis score} = \frac{\textbf{cumulated quarters of bone loss}}{\textbf{total measurable surfaces}}$$

$$\textbf{Caries score} = \frac{\textbf{filled and carious teeth}}{\textbf{total measured teeth}}$$

Due to overlapping and poor contrast in certain regions of the dentition, 21.4% of proximal tooth surfaces in the 800-patient sample were unmeasurable for periodontitis. Similarly in assessing caries experience, 8.2% of the teeth were unmeasurable.

The reliability of the bone loss data was assessed by repeat measurement of 80 duplicate radiographs, selected by random numbering from the 800-patient sample, and the findings are reported in Section 2.3.5. To determine the intra-examiner reproducibility of the DFT data, a further random sample of 80 radiographs (10% of the total) were re-examined. Dahlberg's direct method (Dahlberg, 1940) was used, according to Rugg-Gunn & Holloway (1974), to calculate reliability co-efficients. There was very good agreement both for DFT scores and

measurable teeth: co-efficients of reliability were 0.89 and 0.92 respectively.

4.2.3 Statistical analysis

The Mantel-Haenszel technique (Mantel & Haenszel, 1959) was employed in the statistical analysis of the data to allow for the possible confounders sex, age and teeth present. This test is based on the formulation of multiple 2 x 2 tables relating caries to periodontitis, with the study population grouped into 16 strata, defined by age (4 groups), sex, and 'number of teeth present'. Teeth present, caries and periodontitis groupings were made using the median value for the entire population and dividing the groups into 'above the sample median' and 'below the sample median'. The principles underlying this statistical method have been described in detail by Breslow & Day (1980). The Mantel-Haenszel test was run on an ICL VME mainframe computer using the BMDP statistical package.

4.3 Results

Analysis of the age and sex distribution of the study population indicates that males were, on average, 3 years older than females, and that the age distributions were slightly skewed towards the younger age-groups. The distribution of caries scores by sex showed no differences between males and females.

In order to facilitate further analyses, caries and

periodontitis scores were ranked in order of magnitude and divided into quartiles which were then termed: mild; low to moderate (low—mod); moderate to high (mod—high); and high. A series of cross-tabulations of the grouped scores, performed separately for each age quartile (16—20 years; 21—26 years; 27—37 years; and 38—73 years) were constructed, but gave no suggestion of any association between caries and periodontitis when controlled for age (Figures 4.1—4.4). Thus, Figure 4.1 depicts the relationship between caries and periodontitis in the 16—20-year age-group, and although periodontitis scores are relatively lower than in the other age groups, no pattern emerges for caries experience within the periodontitis severity groupings. A similar lack of pattern in caries and periodontitis experience is seen in the three other age quartiles, although the trend of increasing periodontitis experience with age is clearly evident.

The Mantel-Haenszel analysis, relating periodontitis scores (above/below median) to the caries score (above/below median), stratified for sex (M/F), age (4 categories) and teeth present (above/below median), indicated no relationship between caries and periodontitis ($\chi^2 = 0.00$; $df = 1$; $p > 0.50$). The calculated overall odds ratio for caries and periodontitis is 1.01 with 95% confidence intervals of 0.69 and 1.47. A test for comparison of cross-product ratios within the 16 strata indicated homogeneity within these stratifications ($\chi^2 = 7.19$; $df = 15$; $p = 0.95$)

The Mantel-Haenszel analysis allows for stratification and thus balances the effects of several potential confounders i.e.

Figure 4.1

Cross-tabulation of grouped periodontitis and caries scores for the 16—20-year age quartile.

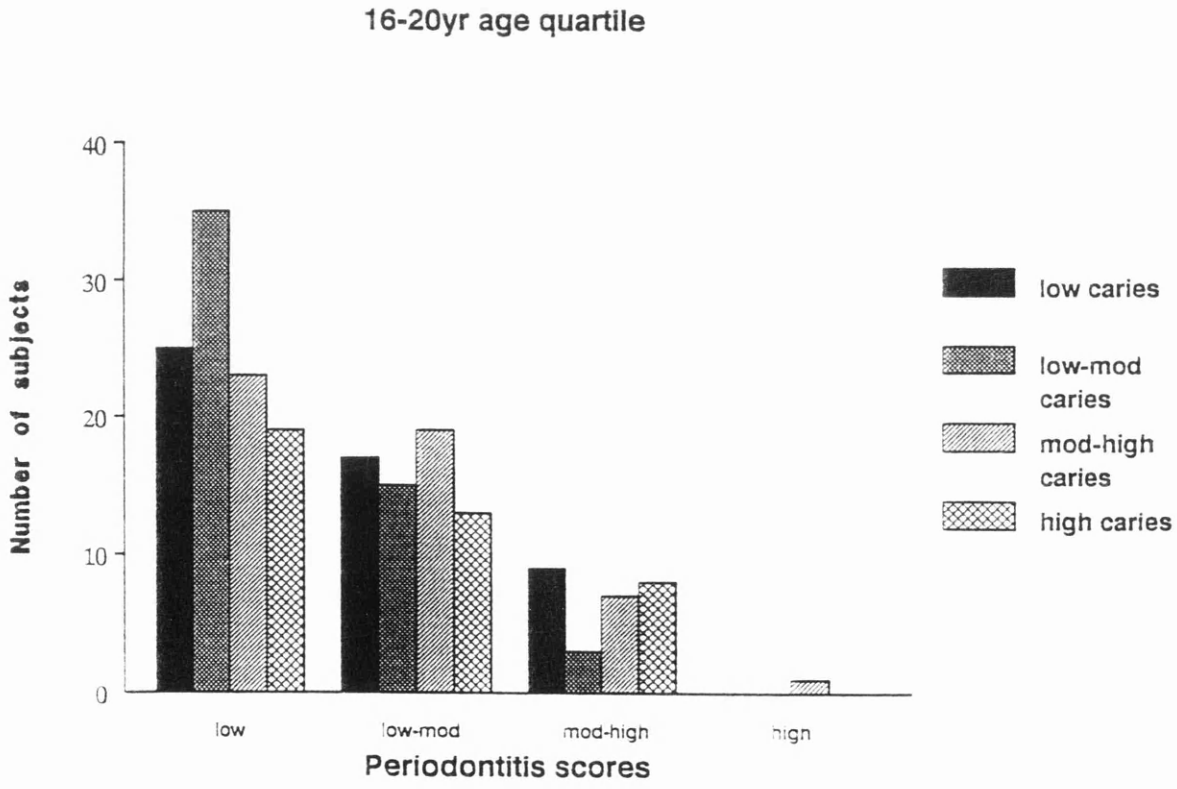


Figure 4.2

Cross-tabulation of grouped periodontitis and caries scores for the 21—26-year age quartile.

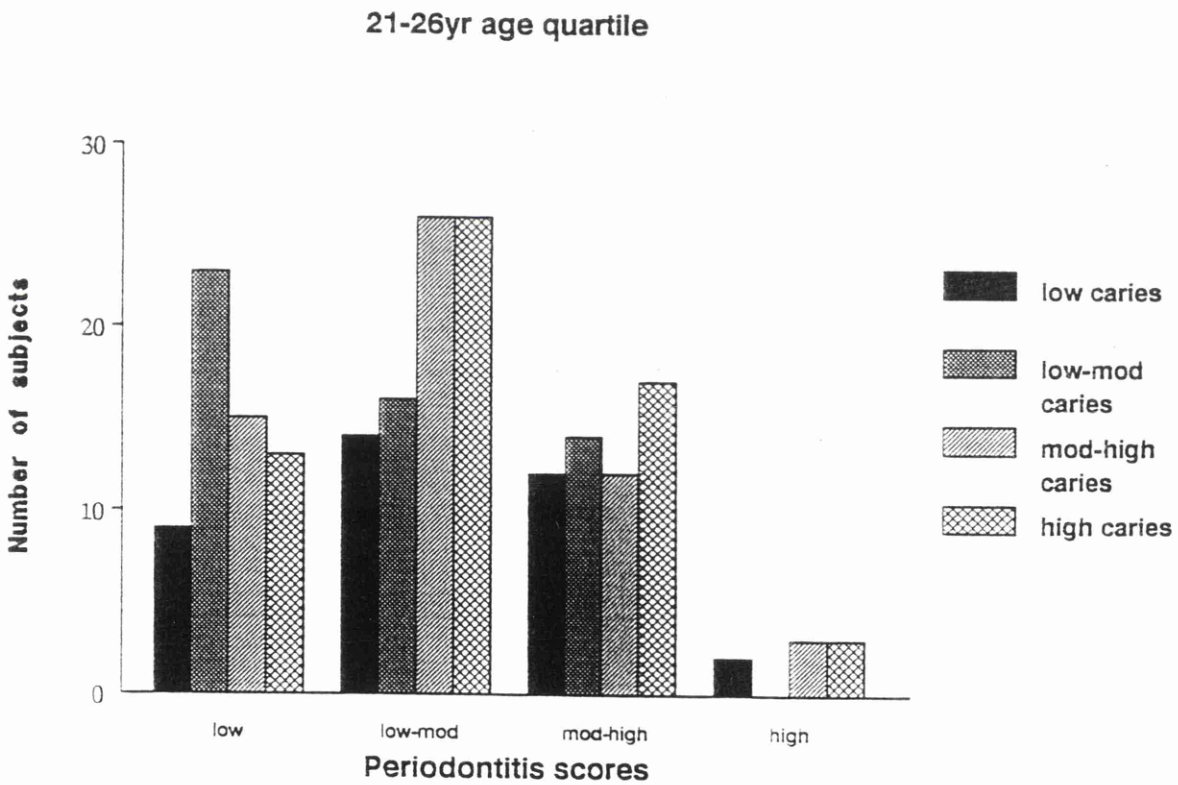


Figure 4.3

Cross-tabulation of grouped periodontitis and caries scores for the 27—37-year age quartile.

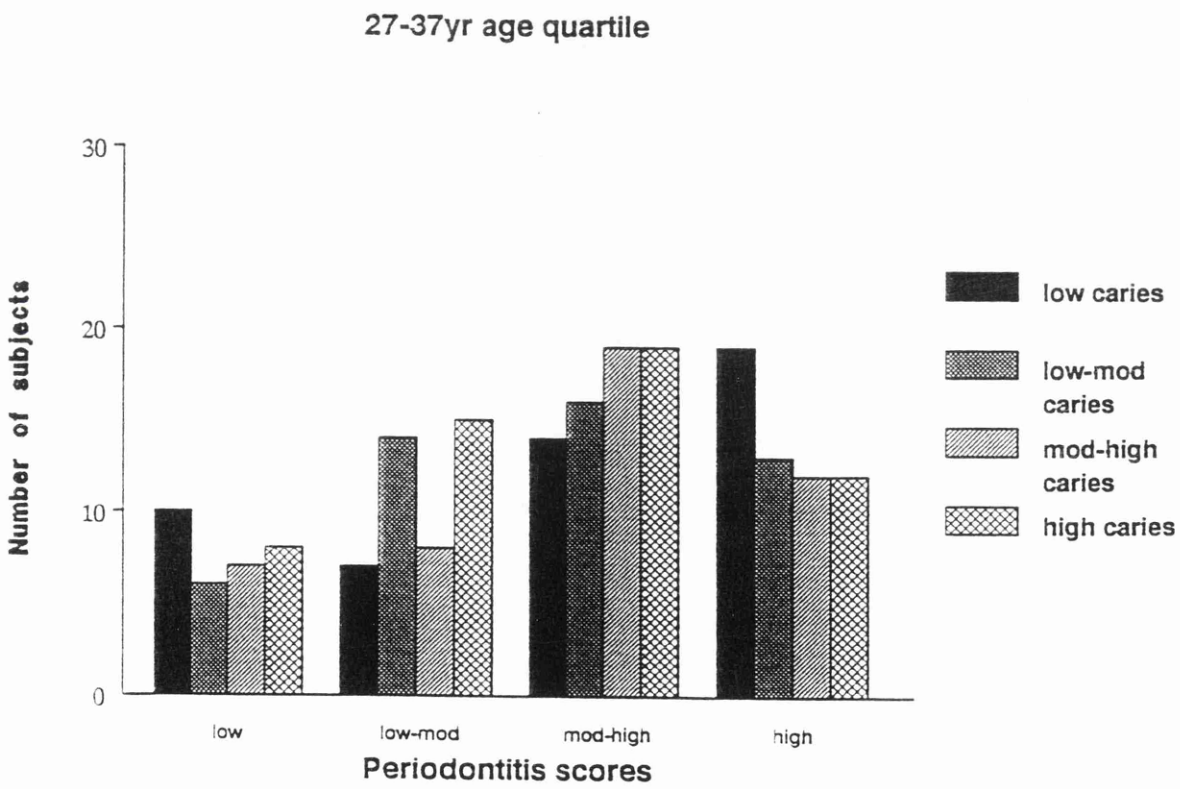
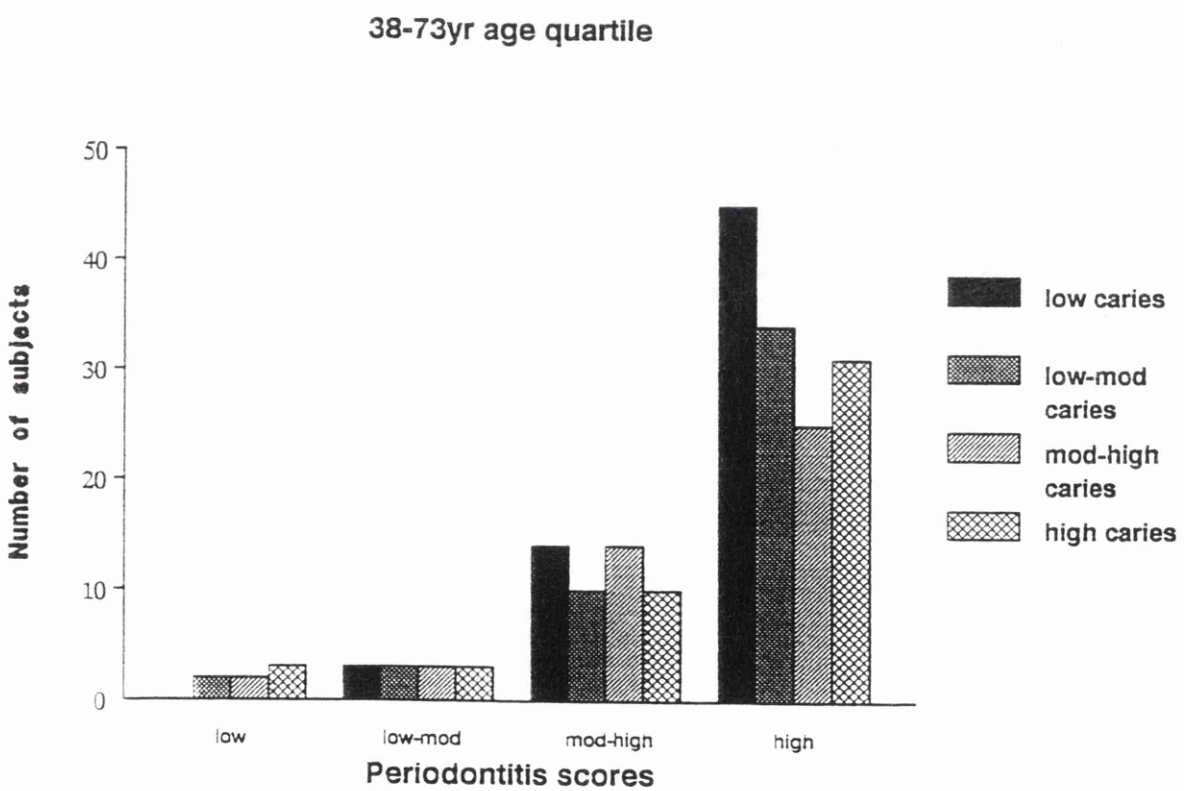


Figure 4.4

Cross-tabulation of grouped periodontitis and caries scores for the 38—73-year age quartile.



age, sex, and number of teeth present, without requiring assumptions to be made about the data. However, it does have the disadvantage that it takes no account of the fact that both caries and periodontitis scores were measured on continuous scales, and so a series of regression analyses were performed to examine the associations between the periodontitis score, caries score, age and sex. The periodontitis scores for males and females were markedly skewed and a log transformation was employed prior to subsequent analysis. Regression analysis revealed a highly significant association between age and periodontal disease (r^2 = 47.3%, $p < 0.001$). A modest but statistically significant association between sex and periodontal score was also found (r^2 = 2.9%, $p < 0.001$), but this association may have resulted from the difference in age distributions for female and male groups. A further analysis was performed to check this and revealed that age and sex were independently related to the periodontitis score. A regression analysis controlling for sex and age was then performed to determine any association between periodontitis and caries. This analysis returned a p value of 0.94 indicating a very clear lack of association between periodontitis and caries. A similar series of analyses to those carried out for the periodontitis index, were performed with caries as the response variable. These analyses showed no association between caries and 'age', 'sex', or 'age and sex' and between caries and periodontitis, controlling for 'age and sex'.

4.4 Discussion

A disadvantage of cross-sectional as compared to longitudinal studies is that missing teeth cannot be fully accounted for, i.e. the reason for extraction is unknown or unreliable. The Mantel-Haenszel technique allows for the possible effect of teeth missing without having to make assumptions about the data. This test enabled stratification of the data such that the possible confounders sex, age and teeth present could be allowed for, and also facilitated presentation. However, the data for periodontitis and caries experience were on continuous scales. Therefore, the association between caries and periodontitis was also assessed using a regression analysis, controlling for age and sex. This more sensitive analysis led to an identical conclusion, that there was no evidence of an association between caries and periodontitis experience.

Thus, these results differ from others where a negative or positive association between caries and periodontitis within the same individual was suggested (Barnett *et al.*, 1986; Fine *et al.*, 1984; Sewõn *et al.*, 1988; Skier & Mandel, 1980). Studies reporting a negative association between caries and periodontitis are largely based on selected patient groups with a recognised increased susceptibility to periodontitis (Barnett *et al.*, 1986; Fine *et al.*, 1984). The only study not using a periodontitis susceptible group which found a negative association was that of Sewõn *et al.* (1988) but this was based on a relatively small sample (115 pairs) from an uncharacterised population. The present study confirms the

tentative conclusion of Ainamo *et al.* (1980), which was made on a young population, and Skier & Mandel (1980) that caries and periodontitis within the same individual are not related.

If there is no association between the risks of caries and periodontitis, we cannot, therefore, predict that individuals with minimal caries should have more or less periodontal destruction than others. Thus, dental primary health care workers should be vigilant in their assessment of both diseases and be wary of the lack of association in the susceptibility to these diseases. Thus, although these common diseases share putative aetiological factors such as oral hygiene practices and dental attendance pattern, the major risk factors are probably quite different. Longitudinal studies in which the reasons for each tooth lost are known would fully test this hypothesis.

CHAPTER 5

LONGITUDINAL STUDY OF UNTREATED PERIODONTITIS

(I) CLINICAL FINDINGS

5.1 Introduction

For many years, a major goal of periodontal research has been the discovery of 'prognostic indicators' which would allow the most susceptible patients and teeth to be singled out for priority treatment. Longitudinal studies during which periodontal status was monitored by traditional clinical means, however, have shown that pre-existing attachment loss, pocket depth, gingival condition, plaque accumulation, suppuration, or bleeding on probing could not be used to predict which sites would subsequently exhibit progressive attachment loss (Listgarten & Levin, 1981; Haffajee, Socransky & Goodson, 1983*a*; Lindhe, Haffajee & Socransky, 1983).

Alternatively, it has been suggested that various specific bacteria or bacterial morphotypes found in samples from periodontal pockets might indicate a susceptibility to destructive periodontitis (Listgarten & Levin, 1981; Claffey *et al.*, 1985; Slots *et al.*, 1985).

The present study was carried out to further elucidate the possible role of clinical and bacteriological criteria in predicting future attachment loss. A further aim was to establish whether the act of collecting subgingival plaque

samples periodically throughout the observation period would have an effect on the clinical or microbiological variables. This chapter is concerned with the clinical findings. The microbiological data are presented in Chapter 6.

5.2 Material and Methods

5.2.1 Subjects

Eleven patients with generalised advanced periodontitis volunteered to take part in this study. Approximately 3 months prior to recruitment, each patient had completed an intensive course of scaling, root planing and oral hygiene instruction with little evidence of clinical improvement: there was no reduction in pocket depths or in the number of bleeding sites, and plaque control remained inadequate. It was, therefore, assumed that this group of patients who were 'resistant' to therapy might be subject to progressive periodontal breakdown and would form a suitable population in which to investigate the possible association between certain clinical and microbiological variables and subsequent attachment loss throughout a 1-year period when no subgingival instrumentation was carried out. The 11 subjects comprised four males and seven females aged 32 to 51 years (mean age 40.7 years). All the participants were in good general health and had not received antibiotic therapy in the previous 6 months.

5.2.2 Selection of study sites

The selection of gingival sites was made at a recruiting appointment before the baseline visit. Bleeding pockets of 4 mm depth or more were selected which were sufficiently accessible for accurate clinical measurements to be made and for bacteriological sampling of subgingival plaque to be carried out. The precise location of each site was determined at the baseline visit and recorded on stone study models which had been obtained from impressions taken at the recruiting visit. A total of 89 teeth, yielding 148 'test' sites, were selected from the left jaw quadrants together with 74 teeth, yielding 117 'control' sites, in the right jaw quadrants.

5.2.3 Experimental design

Figure 5.1 summarises the experimental design. Following their agreement to participate, all subjects were examined on seven occasions at 2-month intervals. With the exception of plaque and gingival redness scores, for which baseline measurements were not obtained until the second visit (due to a last-minute change in protocol), the clinical data were recorded at each visit for each selected site. Bacteriological sampling, however, was carried out at each visit for each selected site only in the left jaw quadrants. The right jaw quadrants were sampled only at the first and last visits. It was intended that left (test) and right (control) jaw quadrants should be compared to assess whether the act of sampling at 2-month intervals had any effect on the clinical or bacteriological variables. During the study, no subgingival instrumentation

Figure 5.1 Experimental design

visit	1	2	3	4	5	6	7
time (months)	0	2	4	6	8	10	12
plaque index		x	x	x	x	x	x
gingival redness		x	x	x	x	x	x
pocket depth	x	x	x	x	x	x	x
attachment level	x	x	x	x	x	x	x
bacteriology (left)	x	x	x	x	x	x	x
bacteriology (right)	x						x

was performed, but scaling and polishing was carried out to remove supragingival calculus and stain, as necessary, from any part of the dentition so affected. All the data collection and bacteriological sampling were carried out by the same individual (the author).

The protocol for this study was approved by the Dental Ethics Committee of Greater Glasgow Health Board, and written informed consent was obtained from each participant.

5.2.4 Plaque index (PlI)

All selected surfaces were scored according to the method of Silness and Löe (1964) on a scale of 0—3.

5.2.5 Gingival redness (GR)

Dichotomous measurements of gingival redness (presence or absence of colour change) were made at each selected site. Bleeding on probing at the pocket orifice was not assessed because of possible interference with subgingival plaque in shallow pockets.

5.2.6 Probing depth (PD)

Measurements were made at each selected site to the nearest millimetre using a Hu-Friedy PCP 12 probe with a tip diameter of 0.4 mm.

5.2.7 Attachment levels

Attachment levels were recorded to the nearest millimetre at each selected site by measuring the distance between the

gingival margin and a fixed reference point on the visible tooth surface (e.g. a cusp tip, a restoration margin or the cemento-enamel junction) and adding this measurement to the corresponding pocket depth. The gingival margin to reference point measurements were made on stone models obtained from alginate impressions taken at each visit. This method was preferred to the construction of an acrylic stent because of obvious occlusal instability among several of the subjects. It was felt that tooth migration and over-eruption would render the stent ill-fitting by the end of the study.

5.2.8 Collection of samples

Supragingival plaque was removed using a curette and discarded. A clean curette was introduced into the pocket as far apically as possible and the bacterial contents removed. The samples were suspended in 0.2 ml sterile anaerobic blood broth and transferred as quickly as possible for laboratory analysis.

5.2.9 Determination of 'improving' or 'deteriorating' sites

Significant changes in attachment levels were identified in two ways.

(1) *During the study*

A loss of attachment of 3 mm or more was taken to be beyond the limit of measurement error and, therefore, reasonably good evidence of loss of periodontal support (Haffajee, Socransky & Goodson, 1983*b*). During the course of the study, all sites exhibiting a loss of attachment of 3 mm or more were given

appropriate treatment in the form of scaling and root planing, and were withdrawn from the study, although the clinical and bacteriological data obtained up to that point were used in the statistical analysis.

(2) *Retrospectively*

At the completion of the study, the sequential changes in probing attachment level at each site were subjected to regression analysis using a method similar to that of Goodson *et al.* (1982). The projected change in attachment level (ΔY) was computed for each site. The ΔY values were used to identify, for each patient, the three sites exhibiting the greatest projected attachment loss and the three sites exhibiting the greatest attachment gain. The mean ΔY was also computed for each patient.

Individual sites were also classified into one of five categories representing the direction and extent of attachment change which had taken place over the one-year study period. To be classified as an 'improving' site the ΔY had to exceed + 1 mm/year. Two grades of 'improving' site were created: a grade I site should exhibit a slope significantly different from 0 at the 5% level; whereas the slope of grade II sites would not differ significantly from zero. Likewise, for a site to be classified as 'deteriorating', the ΔY had to exceed - 1 mm/year. Two grades of 'deteriorating' site were created according to whether the slope of the regression line differed significantly (grade V) from 0 at the 5% level or did not (grade IV). Stable sites (grade III) were those where the ΔY

lay between + 1 and - 1 mm/year and none of these sites had slopes which differed significantly from zero. Thus, a spectrum of attachment change was created ranging from grade I, where there was good evidence of attachment gain, to grade V where there was equally good evidence of attachment loss. This analysis was performed to observe the site distribution of attachment level changes and to determine whether any correlations existed between the clinical and microbiological variables on a site by site basis.

5.2.10 Statistical analysis and data handling

The data for all patients were entered on to a disk file on an ICL 2988 mainframe computer, and the MINITAB statistical package (Ryan, Joiner & Ryan, 1985) was used to analyse the data.

Rank correlation was used to assess whether there was any relationship between mean ΔY values and: (1) the baseline mean PD, PlI and GR scores of each subject; (2) the mean PlI and GR scores of each subject over visits 2—7. Furthermore, the mean values of these variables were compared for the three 'best' and three 'worst' sites of each subject using paired *t*-tests and Wilcoxon signed-ranks tests. This approach allows the subject rather than the site to be taken as the experimental unit (Imrey, 1986).

In addition, the site by site variation was examined by comparing mean levels of PD, PlI and GR for the five attachment change grades using a one-way analysis of variance *F* test, after logarithmic transformation of the data if necessary (Armitage, 1971).

5.3 Results

5.3.1 Attachment level changes

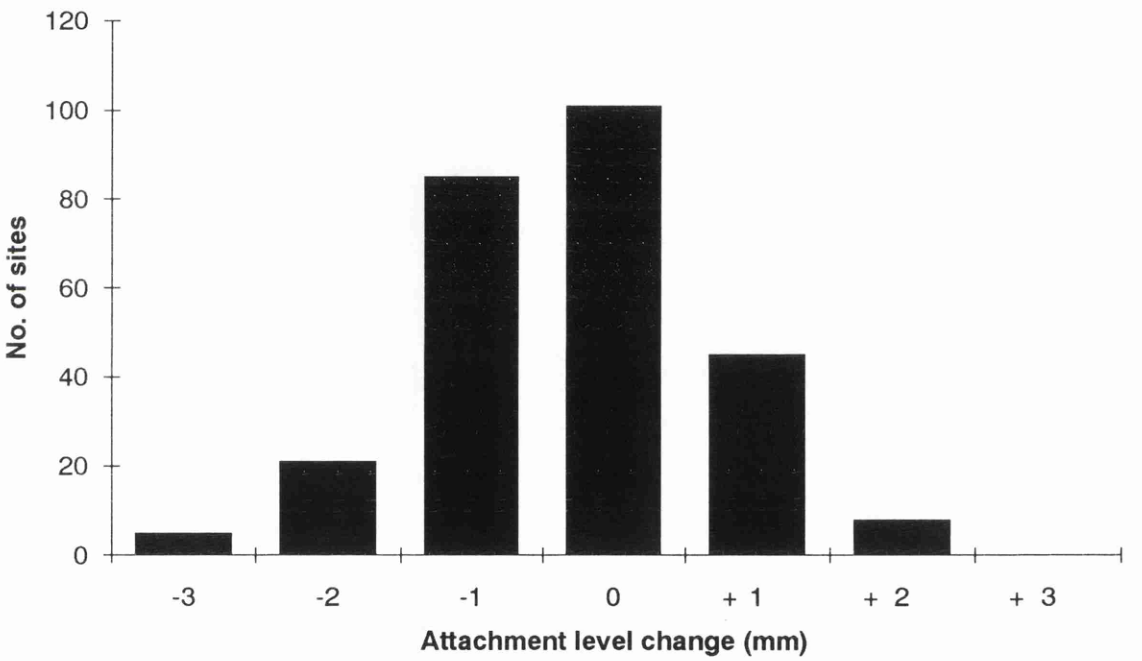
Figure 5.2 shows the distribution of observed changes in attachment level between baseline and final measurement at all sites in all subjects. While there was an observed or apparent gain in attachment of 1 mm or more at a total of 53 sites, there was an apparent loss of attachment of 1 mm or more at a total of 111 sites. The overall picture, therefore, is one of deterioration. During this 1-year observation period, five sites in four subjects lost 3 mm of attachment. Three of these five sites were withdrawn during the study for ethical reasons at the 4-, 8- and 10-month observation periods, while the other two sites did not accumulate 3 mm loss of attachment until the final visit. A further 21 sites in six subjects lost 2 mm of attachment. Therefore, a total of 26 sites in six subjects exhibited attachment loss of 2 mm or more between baseline and final measurement.

These data are reported for descriptive purposes only and to enable comparison with other studies. To allow for measurement error, and obtain a more reliable estimate of disease progress, the whole series of attachment level measurements was subjected to regression analysis. This was thought to be a more sensitive method of identifying attachment changes for subsequent data handling.

Regression analysis yielded a projected change in attachment level (ΔY) for each site studied during the year of observation. On the test side, the results varied from a

Figure 5.2

Observed changes in attachment level between baseline and final measurement



projected attachment loss of 9 mm/year (for the site which was withdrawn at 4 months) to a projected gain of 2.4 mm/year. On the control side, the ΔY varied from a loss of 3.6 mm/year to a gain of 2.4 mm/year. The site distribution of attachment level changes is presented in Table 5.1. The subjects have been ranked according to each individual's overall experience of deterioration or improvement as measured by their overall mean ΔY values. Taking all 265 test and control sites together, 169 (64%) sites were judged to have been stable (grade III) during the year of observation. Improving sites numbered: grade I, 16 (6%); grade II, 15 (6%), while deteriorating sites numbered: grade IV, 33 (12%); grade V, 32 (12%). The frequency and distribution of attachment level changes was similar in both test and control sites.

In comparing the behaviour of different patients' dentitions, it is apparent that the deteriorating sites were fairly widely distributed. Only three subjects, 'A', 'B' and 'C', had no sites in the deteriorating categories. Thirteen of the 31 'improving' sites were found in one subject, 'A', the only subject among the study group who received antibiotic therapy during the study. Penicillin was prescribed by the subject's doctor for a sore throat 1 week before the 8-month observation period and this was thought to explain the improvement in clinical variables which gathered momentum at 10-month and 1-year observation periods.

5.3.2 Reliability of attachment level measurements

As noted above, 169 sites were judged by regression analysis

Table 5.1 No. of sites by grade of attachment level changes

Subject	mean ΔY (mm/year)	Grade I		Grade II		Grade III		Grade IV		Grade V		Total	
		test	control	test	control	test	control	test	control	test	control	test	control
A	+ 0.94	4	4	2	3	8	5	0	0	0	0	14	12
B	+0.35	1	0	0	0	7	8	0	0	0	0	8	8
C	+ 0.32	0	2	1	0	5	8	0	0	0	0	6	10
D	+ 0.02	1	1	2	1	11	8	2	2	1	1	17	13
E	- 0.35	0	0	0	0	8	8	2	0	1	2	11	10
F	- 0.43	1	0	0	0	9	8	1	1	3	2	14	11
G	- 0.47	0	0	0	0	12	8	1	0	2	3	15	11
H	- 0.57	0	0	1	1	6	4	1	1	2	3	10	9
I	- 0.65	0	0	2	0	11	6	2	6	2	1	17	13
K	- 0.70	1	0	1	1	7	9	8	2	3	2	20	14
L	- 0.94	1	0	0	0	9	4	3	1	3	1	16	6
total		9	7	9	6	93	76	20	13	17	15	148	117
total all sites		16		15		169		33		32		265	

Note: subjects ranked A - L in order of increasing tendency towards deterioration

to be stable during the 1-year monitoring period. Standard deviations of the seven repeated attachment level measurements from each stable site ranged from 0.00—0.98 mm with an average standard deviation of 0.45 mm.

5.3.3 PD, PlI and GR as predictors of deterioration

Subjects were ranked according to their mean ΔY value (most improvement = rank 1; most deterioration = rank 11) and also according to their baseline mean PD (shallowest = rank 1), PlI and GR scores (lowest = rank 1). Rank correlation of the mean ΔY value of each subject with baseline mean PD, PlI, and GR scores gave the following results for test and control sites respectively: mean PD (+ 0.01 and - 0.21), mean PlI (+ 0.05 and + 0.04) and mean GR (+ 0.44 and - 0.07). None of these correlation co-efficients are significantly different from zero and there is no evidence of a consistent relationship between the mean amount of deterioration over 12 months and the baseline mean PD, PlI and GR.

To investigate whether real associations were being masked by the inclusion of data from a large number of stable sites, the 'best' three and 'worst' three test and control sites were compared for each subject. Table 5.2 shows the baseline mean PD, PlI and GR scores for the 'best' three and 'worst' three test and control sites of each subject. When these means were compared using paired *t* tests, there were no significant differences between the 'best' sites and the 'worst' sites nor were significant differences to be found between test and control sites. These results were unaltered when Wilcoxon's

Table 5.2 Baseline means of probing depth (PD), plaque index (PII) and gingival redness (GR) for the best 3 sites and the worst 3 sites in each subject

	Test Sites						Control Sites					
	best 3 sites (mean)			worst 3 sites (mean)			best 3 sites (mean)			worst 3 sites (mean)		
Subject	PD (mm)	PII (0 - 3)	GR (0,1)	PD (mm)	PII (0 - 3)	GR (0,1)	PD (mm)	PII (0 - 3)	GR (0,1)	PD (mm)	PII (0 - 3)	GR (0,1)
A	5.0	0.8	1.0	5.7	0.7	1.0	6.7	0.7	1.0	4.3	1.7	1.0
B	5.0	2.3	0.7	5.7	2.0	1.0	6.0	1.7	0.7	5.8	2.3	1.0
C	6.0	1.0	0.3	5.0	1.3	0.3	5.0	1.0	0.8	4.4	1.4	1.0
D	5.7	1.7	0.3	5.7	2.0	1.0	5.0	2.0	1.0	6.3	1.3	1.0
E	5.6	0.4	0.4	5.3	1.0	0.3	5.8	0.5	0.5	5.2	0.5	0.3
F	7.7	1.3	1.0	4.8	2.0	1.0	5.5	1.8	1.0	5.7	1.7	1.0
G	4.3	1.7	1.0	4.7	0.3	1.0	5.3	1.7	1.0	4.7	1.7	1.0
H	4.3	*	*	6.7	*	*	5.3	*	*	4.3	*	*
J	7.0	1.0	0.8	7.7	2.3	1.0	6.0	1.8	0.5	4.8	1.8	1.0
K	4.7	0.7	1.0	4.7	1.0	1.0	5.7	2.0	1.0	4.0	1.7	1.0
L	4.3	1.7	1.0	4.3**	0.8**	0.5**	4.7	0.7	0.7	4.7	0.7	1.0
mean	5.41	1.25	0.75	5.46	1.34	0.82	5.54	1.37	0.81	4.92	1.46	0.93

Paired *t*-test results:

(I) Comparing mean of 'worst' 3 sites with mean of 'best' 3 sites.
(a) Test sites. PD: $t = 0.12$, $p = 0.90$; PII: $t = 0.37$, $p = 0.72$; GR: $t = 0.71$; $p = 0.50$.
(b) Control sites. PD: $t = -2.11$, $p = 0.06$; PII: $t = 0.61$, $p = 0.56$; GR: $t = 1.85$, $p = 0.10$.

(II) Comparing mean of test sites with mean of control sites.
(a) Best 3 sites. PD: $t = -0.35$, $p = 0.73$; PII: $t = -0.57$, $p = 0.58$; GR: $t = -0.66$, $p = 0.53$.
(b) Worst 3 sites. PD: $t = 1.49$, $p = 0.17$; PII: $t = -0.53$, $p = 0.61$; GR: $t = -1.48$, $p = 0.17$.

* Missing data
** Mean values given for worst 4 sites, 2 of which tied for 3rd place.

signed-ranks test was used instead of the paired *t*-test.

This lack of association was confirmed when the baseline mean levels of PD, PlI and GR were compared for the five attachment change grades of test and control sites, treating the sites as independent units (data not shown).

Thus attachment level changes were not dependent on *initial* pocket depths, plaque or gingival redness. There were, furthermore, no appreciable differences between test and control sites with respect to initial pocket depths, plaque or gingival redness.

5.3.4 PlI and GR over visits 2—7

Rank correlation of the mean ΔY value of each subject with mean PlI and GR scores over visits 2—7 gave the following results for test and control sites respectively: mean PlI (- 0.08 and + 0.38) and mean GR (+ 0.46 and + 0.27). Although there is a suggestion of correlation here, between GR and loss of attachment, none of these correlations are statistically significant (e.g. rank correlation = + 0.46, $n = 10$, $p = 0.17$).

Table 5.3 shows the mean PlI and GR scores over visits 2—7 for the 'best' three and 'worst' three test and control sites of each subject. When these means were compared using paired *t*-tests, there were no significant differences between the 'best' sites and the 'worst' sites, nor were significant differences observed between test and control sites. These results remained unaltered when Wilcoxon's signed-ranks test was used instead of the paired *t*-test.

This lack of association was confirmed when the mean PlI

Table 5.3 Mean plaque index (PII) and gingival redness (GR) scores over visits 2 - 7 for the best 3 sites and the worst 3 sites in each subject

	Test Sites				Control sites			
	best 3 sites (mean)		worst 3 sites (mean)		best 3 sites (mean)		worst 3 sites (mean)	
Subject	PII (0 - 3)	GR (0.1)	PII (0 - 3)	GR (0.1)	PII (0 - 3)	GR (0.1)	PII (0 - 3)	GR (0.1)
A	1.0	0.6	0.4	0.4	0.7	0.8	1.6	0.7
B	2.1	0.9	2.1	0.8	1.6	0.6	1.8	0.9
C	1.1	0.5	0.8	0.8	0.6	0.8	1.0	0.7
D	0.8	0.2	1.8	0.8	1.3	0.4	1.5	0.9
E	0.6	0.2	1.2	0.3	0.4	0.1	0.5	0.1
F	1.5	0.9	1.3	1.0	1.8	1.0	2.0	0.9
G	1.4	0.8	1.0	0.9	1.8	0.9	1.7	1.0
H	*	*	*	*	*	*	*	*
J	1.2	0.8	2.6	0.9	1.7	0.7	1.5	0.6
K	0.3	1.0	1.1	0.8	1.8	1.0	1.5	1.0
L	1.4	0.9	0.6	0.7	1.1	0.6	0.8	0.9
mean	1.15	0.67	1.28	0.74	1.26	0.70	1.39	0.78

Paired *t*-test results:
(I) Comparing mean of 'worst' 3 sites with mean of 'best' 3 sites.
(a) Test sites. PII: $t = 0.58$, $p = 0.57$; GR: $t = 0.84$, $p = 0.42$.
(b) Control sites. PII: $t = 1.08$, $p = 0.31$; GR: $t = 1.06$, $p = 0.32$.

(II) Comparing mean of test sites with mean of control sites:
(a) Best 3 sites. PII: $t = -0.61$, $p = 0.65$; GR: $t = -0.65$, $p = 0.53$.
(b) Worst 3 sites. PII: $t = -0.46$, $p = 0.65$; GR: $t = -0.65$, $p = 0.53$.

* Missing data.

and GR scores over visits 2—7 were compared for the five attachment change grades of test and control sites, treating the sites as independent units (data not shown).

Thus attachment level changes were not dependent on the *prevailing* level of plaque or gingival redness throughout the observation period. Furthermore, the act of sampling carried out at test sites on each occasion appeared to have no effect on plaque or gingival redness scores.

5.3.5 PlI and GR in each subject at each visit

Tables 5.4 and 5.5 show the mean PlI and GR scores respectively for all sites in each subject at each visit. The slopes of the least squares linear regression lines of mean PlI and mean GR scores on time have been computed for each subject. The subjects have been ranked according to these slopes and rank correlation was used to compare these rankings with the subjects' rankings on clinical improvement/deterioration. The rank correlation co-efficients were + 0.36 for PlI ($p = 0.25$) and + 0.53 for GR ($p = 0.09$). If subject 'A' is excluded, the rank correlation for GR falls to + 0.37 ($p = 0.26$). This subject, it must be remembered, finished a course of penicillin 1 week before the 8-month observation period at which point his mean GR scores fell markedly and had not risen by the 10-month and 1-year observation periods.

Thus, with the exception of subject 'A', those subjects who suffered the greatest amounts of attachment loss during the 1-year of observation did not show a tendency towards greater mean PlI or GR values with time, nor did patients with a

Table 5.4 Mean plaque index values from 2 - 12 months for individual subjects

Subject	Months						Rank by PII 'improvement'
	2	4	6	8	10	12	
A	1.0	1.0	1.1	0.9	1.1	1.3	9
B	2.1	1.7	1.9	1.8	2.0	1.8	4.5
C	1.3	0.9	0.9	0.6	0.9	0.5	1
D	1.7	1.6	1.0	0.9	1.5	1.3	2
E	0.6	0.4	0.9	0.3	1.1	0.7	10
F	*	*	1.3	1.0	1.4	1.1	4.5
G	1.5	1.2	1.1	0.9	1.0	1.2	3
H	1.1	0.9	1.4	1.2	1.0	1.0	6
J	1.5	1.9	1.5	1.4	1.7	1.8	7
K	1.6	1.8	1.7	1.6	1.8	2.1	11
L	1.8	1.4	1.3	1.3	1.7	1.8	8

* Missing data

Table 5.5 Mean gingival redness scores from 2 - 12 months for individual subjects

Subject	Months						Rank by GR 'improvement'
	2	4	6	8	10	12	
A	0.96	0.65	0.85	0.50	0.50	0.31	1
B	0.81	0.81	0.75	0.69	0.94	0.81	9
C	0.69	0.75	0.56	0.75	0.63	0.50	3
D	0.80	0.70	0.63	0.43	0.70	0.60	2
E	0.38	0.05	0.14	0.19	0.33	0.00	5.5
F	*	*	0.79	0.79	0.79	0.84	10
G	1.00	0.91	0.94	0.79	0.91	0.82	4
H	0.77	0.73	0.96	0.71	0.81	0.57	5.5
J	1.00	0.84	0.81	0.69	0.92	0.92	8
K	0.83	0.47	0.41	0.74	0.71	0.79	11
L	1.00	0.96	0.96	0.92	0.88	0.96	7

* Missing data

tendency towards clinical improvement show any trend towards lower mean PlI and GR scores.

5.4 Discussion

The main purpose of the present study was to investigate whether periodontal breakdown might be related to pre-existing or prevailing clinical conditions or bacteriological findings. To test this hypothesis, it was important that the population studied should exhibit a sufficient incidence of clinically detectable ongoing disease.

By the 1-year observation period, five out of 265 sites had demonstrated loss of attachment of *more than 2 mm*, a rate of 1.9% of sites per year. By comparison, Lindhe *et al.* (1983) found that 1.9% of sites per year exhibited attachment loss of *more than 2 mm* in a 6-year study of 64 untreated Swedish subjects with mild to moderate periodontitis. The same authors reported that 3.2% of sites exhibited attachment loss of this same magnitude in a 1-year study of 36 untreated Americans with advanced periodontitis (Lindhe *et al.*, 1983).

Taking as a less stringent criterion of periodontal breakdown, an observed loss of attachment of *2 mm or more*, 26 out of 265 sites in the present study fell into that category, a rate of 9.8% of sites per year. Corresponding annual rates of breakdown of *2 mm or more* in the above mentioned studies were 6.2% for Swedish subjects (Lindhe *et al.*, 1983) and 9.1% for American subjects (Lindhe *et al.*, 1983). In addition, Lang

et al. (1986) found that only 3.1% of sites per year exhibited attachment loss of *2 mm or more* in a 2-year study of 1054 sites in 55 patients under recall maintenance following treatment of advanced periodontitis.

Thus, the incidence of periodontal breakdown reported in the present study is similar to, and in some cases greater than the incidence in other study populations.

Linear regression analysis was the method chosen in the present study to identify 'improving' and 'deteriorating' sites (Goodson *et al.*, 1982; Badersten, Nilvéus & Egelberg, 1985) so that the relationship, if any, between disease 'activity' and other clinical and bacteriological variables could be investigated. This method allows the rate of change of attachment (the slope of the regression line) to be evaluated in conjunction with the variability of the longitudinal measurements as determined by the probability level of the slope. In the present study, grade I and grade V sites had a greater validity as 'improving' and 'deteriorating' sites respectively than grade II and grade IV sites since the former sites not only exhibited a projected attachment change of more than 1 mm per year but their slopes also differed significantly from zero at the 5 per cent level.

The reproducibility of attachment level measurements was assessed by calculating standard deviation values for the seven repeated attachment level measurements from each stable site. This gave an average standard deviation of 0.45 mm within a range of 0.00 mm to 0.98 mm, suggesting that, since probing measurements are normally distributed (Glavind & Löe, 1967),

a difference of 2 mm between two *single* measurements would be sufficient to detect significant changes at the $p < 0.05$ level. This represents an improvement in overall detection sensitivity when compared with data presented by Goodson *et al.* (1982). These authors obtained standard deviation values of 0.60—1.41 mm with an overall mean of 0.84 mm from monthly attachment level measurements of 635 sites, judged by regression analysis to have been stable during the 1-year monitoring period. The comparatively high level of reproducibility in the present study may be partly attributed to the exclusion of all sites where access for probing was difficult.

Much has been written recently concerning the use of site or subject as the experimental unit in periodontal research (Blomqvist, 1985; Laster, 1985; Imrey, 1986). The general consensus favours the use of the subject as experimental unit, since sites within subjects cannot be regarded as independent. In this study, both approaches to analysis were used and the results obtained are very similar. This will not, of course, be universally true.

It might have been expected that the act of sampling subgingival plaque every 2 months during the one year of observation would have had a beneficial effect on the periodontium of the test sites. This proved not to have been the case at all, there being no major difference in the frequency of attachment level changes or in mean plaque or gingival redness scores between test sites which were sampled at each visit and control sites which were sampled at the first and last visit only. By contrast, Mousques, Listgarten &

Stoller (1980), investigating the effect of sampling on periodontal conditions in 18 subjects, observed a trend towards lower plaque and gingival index scores and slightly shallower pocket depths, 42 days after the pockets were sampled. The authors, however, were inclined to attribute this to the subjects' improved awareness of oral hygiene rather than an effect of sampling itself.

The finding that attachment level changes were not predicted by *initial* plaque, gingival redness, or pocket depth scores is entirely in accordance with other reports (Haffajee *et al.*, 1983*a*; Listgarten & Levin, 1981; Lindhe *et al.*, 1983). Furthermore, in this study, attachment loss occurred independently of the *prevailing* oral hygiene and gingival condition throughout the observation period. Although there is a suggestion of correlation between the prevailing level of gingival redness and deterioration in attachment levels, when results are averaged over all sites for each subject, the correlations observed are not statistically significant. Furthermore, there is no association between prevailing levels of gingival redness and attachment change when the 'best' three sites are compared with the 'worst' three sites for each patient. Also, none of the significance levels quoted have been corrected to allow for the fact that multiple significance tests have been performed. Such a correction would considerably increase the *p*-values quoted and lessen the statistical significance.

While slight variations in plaque and gingivitis were recorded in each subject between visits, there was no

consistent upward or downward trend, either in subjects who showed an overall improvement in attachment levels, or in those who suffered attachment loss.

In conclusion, this study of untreated periodontitis supports the findings of other workers that plaque, gingival inflammation and pocket depth measurements cannot be used in a predictive capacity with respect to attachment loss. Furthermore, the act of bacteriological sampling at 2-month intervals for one year had no effect on the progress of periodontal disease.

CHAPTER 6

LONGITUDINAL STUDY OF UNTREATED PERIODONTITIS (II) MICROBIOLOGICAL FINDINGS

6.1 Introduction

It is now well-established that traditional clinical criteria are inadequate for determining active disease sites in periodontitis, or for measuring the degree of susceptibility to future disease (Listgarten & Levin, 1981; Haffajee *et al.*, 1983*b*; Lindhe *et al.*, 1983). Since bacteria are involved in the aetiology of most, if not all forms of periodontal disease, a number of workers have investigated the use of microbiological markers to assist in the diagnosis and clinical management of chronic periodontitis.

6.1.1 Dark-field microscopy

Dark-field microscopical techniques have been used most commonly, and the use of these tests in periodontitis has been reviewed by Greenstein & Polson (1985). Generally, spirochaetes have been found more frequently, and in higher numbers at diseased sites, compared to healthy sites (Listgarten & Helldén, 1978; Lindhe, Liljenberg & Listgarten, 1980; Armitage *et al.*, 1982). In addition, attempts have been made to correlate spirochaete counts with a number of clinical variables; the strongest relationship being between spirochaete

counts and pocket depth (Rosenberg, Evian & Listgarten, 1981; Evian, Rosenberg & Listgarten, 1982). In a longitudinal study, Listgarten & Levin (1981) showed that spirochaete counts could be used to predict which subjects would experience active periodontitis in the following 12 months.

6.1.2 Culture studies

Culture studies have also been used to assist in clinical diagnosis, and a close relationship between percentages of *Bacteroides gingivalis*[1], *Bacteroides intermedius*[2] and *Actinobacillus actinomycetemcomitans* and disease-active periodontitis has been reported (Slots *et al.*, 1985, 1986). However, no such correlations were found by Moore *et al.*, (1983).

6.1.3 Aims

Due to the variability in rate of attachment loss in chronic periodontitis, longitudinal studies are more appropriate than cross-sectional investigations for relating microorganisms to disease activity. In addition, during the period of study, ideally, patients should receive no therapy. However, most previous microbiological investigations have been cross-sectional, and, in the few longitudinal studies which have been performed, usually treatment of some kind has formed part of the experimental protocol. Therefore, the present longitudinal study was carried out to relate changes in probing attachment

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[1]Now reclassified to *Porphyromonas gingivalis*

[2]Now reclassified to *Prevotella intermedia*

level to the % spirochaetes and % black-pigmented *Bacteroides* species in samples collected over a 1-year period from untreated patients with chronic periodontitis. A further aim was to establish whether the act of collecting subgingival plaque samples periodically throughout the observation period would have an effect on the microbiological findings.

6.2 Material and Methods

6.2.1 Subjects and experimental design

The subjects investigated and the experimental design of the longitudinal study are described in Section 5.2. Eleven subjects with generalised advanced periodontitis volunteered to take part in the 1-year study. All the participants were in good general health and had not received antibiotic therapy during the last 6 months. Bacteriological samples were collected at each of the seven visits from pre-selected sites in the left jaw quadrants (test sites), and on the first and last visits only from the sites in the right jaw quadrants (control sites). The determination of 'improving' or 'deteriorating' sites, both during the study and retrospectively, was performed as described in Section 5.2.9.

6.2.2 Collection of samples

All the bacteriological sampling was carried out by the same individual (the author). Supragingival plaque was removed, using a curette, and discarded. A clean curette was introduced

into the pocket, as far apically as possible, and the bacterial contents removed. The samples were suspended in 0.2 ml of sterile anaerobic blood broth (Gibco—Paisley, Scotland) by vigorously agitating the tip of the instrument in the fluid. Plaque samples were transferred to the laboratory as quickly as possible, and disaggregated by aspirating and expelling the fluid 10—20 times through a disposable tuberculin syringe with a 25-gauge needle.

6.2.3 Microscopy and counting

The method used for counting the percentage of spirochaetes in subgingival plaque was that of MacFarlane, McCourtie & Watkins (1986). This technique involved the combined use of negatively stained smears, dark-field microscopy and an image analysing system. Smears were prepared by adding 2 μl of the plaque suspension and 4 μl of filter-sterilised 10% Nigrosin (BDH Chemicals Ltd., Poole, England) towards one end of a previously cleaned glass slide. The solutions were mixed and then evenly smeared over about two thirds of the glass slide using the edge of a 22 mm glass cover slip. The smears were dried in air for 15 minutes then mounted in one drop of Harleco Synthetic Resin (Kodak Chemicals, Liverpool, England) with a 22 mm square coverslip.

The smears were examined on a Leitz Ortholux microscope with a 12 v 100 w power pack, using a darkground condensor (D 0.80 0.95) and a 630 x dry fluorite objective containing a D 0.80 funnel stop. A Newvicon television scanner was connected to the phototube of the microscope and to an image analysing

Optomax III System (Analytical Measuring Systems, Saffron Walden, Essex, England). The field under study was displayed on a video monitor, the microorganisms appearing white on a black background. After setting the detector control, the total bacterial count for each field was obtained in seconds using the 'total count' facility on the Optomax III System. It was possible to check by eye what the image analyser was counting, since a white 'flag' appeared on the television monitor screen next to each object being enumerated. Therefore, the operator uses his own visual acuity as the standard to which the machine is adjusted, and is able to monitor the level of non-bacterial particulate matter in each field. Fields which contained obvious particulate debris were not counted.

Since the analyser could not differentiate between spirochaetes and other bacteria, it was necessary to count the number of spirochaetes by eye and to record these for each field. Duplicate smears were prepared from each sample, and 30 fields per smear were counted. The Optomax III System was interfaced with an Apple II Europlus Computer, allowing the results from each field to be stored on a floppy disc and printed on an Epson printer MX80F/T III (Sinshu Seiki Co. Ltd., Nagano, Japan).

The remainder of the sample was diluted 1:100 and 1:1000 in 1 ml volumes of sterile anaerobic blood broth (Gibco—Paisley, Scotland). Using a spiral plating machine (Don Whitley Scientific, Shipley, England), 50 μl of each sample were inoculated onto a brain heart infusion agar plate

(Gibco—Paisley, Scotland) supplemented with 7.5% blood and 1 ml vitamin K/haemin solution (Gibco—Paisley, Scotland). Plates were incubated for 7 days at 37°C in an atmosphere of 85% N_2, 10% H_2 and 5% CO_2 within an anaerobic chamber (Forma Scientific, Marietta, Ohio).

After incubation, using a Spiral System Counting Grid, (Don Whitley Scientific, Shipley, England) the total viable count and the number of black-pigmented bacteroides colonies, present in each sample, were counted. Wherever possible, the whole plate was counted, but, where this was not possible, a segment of the plate was used. In order to balance any irregularities in sample deposition, a similar segment on another part of the plate was also counted.

6.2.4 Statistical analysis and data handling

The data for all subjects were entered on to a disk file on an ICL 2988 mainframe computer, and the MINITAB statistical package (Ryan *et al.*, 1985) was used to analyse the data. Rank correlation was used to assess whether there was any relationship between mean ΔY values (projected changes in attachment level) and: (1) the baseline mean % spirochaete or % black-pigmented bacteroides counts of each subject; (2) the mean % spirochaete or % black-pigmented bacteroides counts of each subject at the 12-month visit; (3) the mean % spirochaete and % black-pigmented bacteroides scores of the test sites of each subject over visits 1—7. Furthermore, the mean values of these variables were compared for the three 'best' and three 'worst' sites of each subject using paired *t*-tests and Wilcoxon

signed-ranks tests. This approach allows the subject rather than the site to be taken as the experimental unit (Imrey, 1986). In addition, the site-by-site variation was examined by comparing mean levels of % spirochaetes and % black-pigmented bacteroides for the five attachment change grades, defined below, using a one-way analysis of variance *F* test, after logarithmic transformation of the data if necessary (Armitage, 1971).

6.3 Results

6.3.1 Attachment level changes

A total of 148 test and 117 control sites were examined in the 11 subjects. Taking all 265 test and control sites together, 169 (64%) sites were stable (grade III during the year of observation), 31 (12%) improved (16 grade I and 15 grade II), while deteriorating sites numbered 65 (24%) (33 grade IV and 32 grade V). The frequency and distribution of attachment level changes were similar in both test and control sites. Before the end of the study, three teeth (two on the test side and one on the control side) exhibited loss of attachment of 3 mm and were withdrawn for treatment.

6.3.2 Percentage spirochaetes and percentage black-pigmented bacteroides as predictors of deterioration

Subjects were ranked according to their mean ΔY value (most improvement = rank I; most deterioration = rank II), and also

according to their baseline mean % spirochaetes and black-pigmented bacteroides (lowest value = rank I). Rank correlation of the mean ΔY value of each subject with baseline mean % spirochaetes and % black-pigmented bacteroides scores gave the following results for test and control sites respectively: mean % spirochaetes (+ 0.22 and + 0.39); mean % black-pigmented bacteroides (- 0.11 and - 0.03). None of these correlation co-efficients are significantly different from zero and there is no evidence of a consistent relationship between the mean amount of deterioration over 12 months and the baseline mean microbiological values.

To investigate whether real associations were being masked by the inclusion of data from a large number of stable sites, the 'best' three and 'worst' three test and control sites were compared for each subject. Table 6.1 shows the baseline mean % spirochaetes and % black-pigmented bacteroides scores for the 'best' three and 'worst' three test and control sites of each subject. When these means were compared using paired *t*-tests, there were no significant differences between the 'best' sites and the 'worst' sites nor were significant differences to be found between test and control sites. These results were unaltered when Wilcoxon's signed-ranks test was used instead of the paired *t*-test.

This lack of association was confirmed when the baseline mean levels of % spirochaetes and % black-pigmented bacteroides were compared for the five attachment change grades of test and control sites, treating the sites as independent units (data not shown).

Table 6.1 Baseline values of % spirochaetes and % black pigmented bacteroides (BPB)

	Test sites				Control sites			
	best 3 sites		worst 3 sites		best 3 sites		worst 3 sites	
Patient	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB
A	2.3	0.0	1.4	0.0	12.5	0.0	8.4	0.0
B	1.4	4.0	1.8	17.6	3.2	5.4	4.4	11.2
C	9.2	0.0	1.1	4.5	1.4	0.7	3.7	17.2
D	7.5	29.2	12.9	54.3	15.4	52.2	9.3	43.2
E	4.6	0.9	4.6	0.1	8.0	0.0	2.1	0.1
F	20.4	*	23.3	*	12.6	*	11.8	*
G	1.8	0.0	5.0	12.2	6.2	7.9	5.6	7.6
H	6.0	0.1	6.0	0.1	5.5	1.7	6.7	2.0
J	11.9	6.8	6.1	4.9	5.8	0.6	7.8	1.5
K	9.9	0.1	11.4	0.5	14.5	0.4	9.4	4.6
L	1.2	0.1	3.0	1.5	7.2	4.2	4.5	0.2
mean	6.93	4.12	6.96	9.57	8.39	7.31	6.70	8.76

Paired *t*-test results:

(I) Comparing mean of 'worst' 3 sites with mean of 'best' 3 sites.
(a) Test sites. SPIRO: $t = 0.03$, $p = 0.98$; BPB: $t = 1.96$, $p = 0.08$.
(b) Control sites. SPIRO: $t = -1.74$, $p = 0.11$; BPB: $t = 0.69$, $p = 0.51$.

(II) Comparing mean of 'test' sites with mean of 'control' sites.
(a) 'Best' 3 sites. SPIRO: $t = -0.77$, $p = 0.46$; BPB: $t = -1.29$, $p = 0.23$.
(b) 'Worst' 3 sites. SPIRO: $t = 0.19$, $p = 0.86$; BPB: $t = 0.40$, $p = 0.70$.

* Missing data

Thus, attachment level changes were not dependent on the *initial* % spirochaete or % black-pigmented bacteroides counts. There was, furthermore, no appreciable difference between test and control sites with respect to the microbiological data.

6.3.3 Percentage spirochaetes and percentage black-pigmented bacteroides at the 12-month visit

Rank correlation of the mean ΔY value of each subject with mean % spirochaetes and % black-pigmented bacteroides scores at visit 7 gave the following results for test and control sites respectively: mean % spirochaetes (+ 0.64 and + 0.88) and mean % black-pigmented bacteroides (+ 0.04 and - 0.28). There was a significant correlation at visit 7 between low mean spirochaete levels and mean ΔY values for both the control (p = 0.006) and test (p = 0.04) sites. However, none of the correlations for the % black-pigmented bacteroides counts were statistically significant.

Table 6.2 shows the mean microbiological scores at the 12-month visit for the 'best' three and 'worst' three test and control sites of each subject. When these means were compared using paired t-tests, there were no significant differences between the 'best' sites and the 'worst' sites, nor were significant differences observed between test and control sites. These results remained unaltered when Wilcoxon's signed ranks test was used instead of the paired t-test.

This lack of association was confirmed when the mean % spirochaete and % black-pigmented bacteroides scores at the 12-month visit were compared for the five attachment change grades

Table 6.2 % spirochaetes and % black pigmented bacteroides (BPB) at the 12-month visit.

	Test sites				Control sites			
	best 3 sites		worst 3 sites		best 3 sites		worst 3 sites	
Patient	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB
A	0.0	2.5	3.9	16.5	0.9	6.2	0.6	3.7
B	0.0	9.5	1.2	1.8	2.3	6.2	3.8	18.8
C	15.2	0.3	5.5	0.0	7.5	0.1	5.0	0.0
D	0.0	24.7	1.9	28.5	3.3	39.0	4.6	44.5
E	2.2	0.0	0.8	11.0	0.0	0.2	2.4	1.0
F	27.1	1.3	63.1	3.7	43.4	0.4	40.4	7.8
G	1.8	3.2	3.5	1.1	6.0	8.3	6.9	5.0
H	9.9	3.9	5.0	14.9	5.0	10.2	9.7	7.1
J	13.8	4.1	19.7	5.5	13.7	2.3	10.8	5.3
K	1.7	7.9	17.3	7.1	5.2	4.5	16.8	3.6
L	14.5	0.2	11.1	0.8	12.3	0.2	10.0	1.3
mean	7.84	5.24	12.09	8.26	9.05	7.05	10.09	8.92

Paired *t*-test results:

(I) Comparing mean of 'worst' 3 sites with mean of 'best' 3 sites.

(a) Test sites. SPIRO: $t = 1.14$, $p = 0.28$; BPB: $t = 1.54$, $p = 0.15$.

(b) Control sites. SPIRO: $t = 0.80$, $p = 0.44$; BPB: $t = 1.25$, $p = 0.24$.

(II) Comparing mean of 'test' sites with mean of 'control' sites.

(a) 'Best' sites. SPIRO: $t = -0.65$, $p = 0.53$; BPB: $t = -1.15$, $p = 0.28$.

(b) 'Worst' sites. SPIRO: $t = 0.85$, $p = 0.42$; BPB: $t = -0.23$, $p = 0.82$.

of test and control sites, treating the sites as independent units (data not shown). Therefore, although rank correlation of the data yielded a significant relationship between mean spirochaete counts and the subjects with greatest mean improvement in attachment levels, this result was not evident when the three 'worst' and three 'best' sites were compared, or when the microbiological data were analysed on a site-specific basis.

6.3.4 Percentage spirochaetes and percentage black-pigmented bacteroides throughout the 1 year of observation

Rank correlation of the mean ΔY value of each subject with the mean % spirochaetes and mean % black-pigmented bacteroides scores, accumulated at test sites over all seven visits, produced correlation co-efficients of + 0.24 with mean % spirochaetes and + 0.12 with mean % black-pigmented bacteroides. When the mean % spirochaetes and mean % black-pigmented bacteroides of the 'best' three and the 'worst' three test sites were compared for each subject (Table 6.3), no significant differences were found.

This lack of association was confirmed when the mean levels of % spirochaetes and % black-pigmented bacteroides obtained from the test sites over all seven visits were compared for the five attachment change grades, i.e., analysing each site as an independent unit. However, a slight but not quite significant trend towards higher mean spirochaete counts in deteriorating sites was found.

Table 6.3 % spirochaetes and % black pigmented bacteroides (BPB) for all test site data accumulated during the 12-month observation period.

	Best 3 sites		Worst 3 sites	
Patient	mean % spiro	mean % BPB	mean % spiro	mean % BPB
A	4.7	5.2	6.8	4.4
B	6.3	1.4	5.2	11.8
C	7.0	16.7	15.2	19.9
D	10.9	1.7	2.9	3.6
E	3.0	2.0	2.4	3.8
F	21.6	*	36.1	*
G	1.2	4.5	6.9	4.2
H	5.1	5.8	3.9	3.7
J	16.3	4.1	11.5	3.3
K	9.3	5.4	14.3	7.8
L	9.4	11.5	7.8	14.7
mean	8.62	5.83	10.27	7.72

Paired *t*-test results.
Comparing mean of 'worst' 3 sites with mean of 'best' 3 sites.
SPIRO: $t = 0.87$, $p = 0.41$; BPB: $t = 1.70$, $p = 0.12$.

* Missing data.

6.3.5 Means of percentage spirochaetes and percentage black-pigmented bacteroides for each subject

Table 6.4 shows the mean % spirochaetes at test sites for each subject at each visit. There was no evidence of a relationship between baseline mean % spirochaetes and each subject's subsequent experience of attachment loss. Furthermore, although there was considerable inter-subject variation in % spirochaetes, there was some evidence of consistency in individual mean % spirochaete counts over the 1-year period of observation. An exception was subject 'A' who received penicillin therapy just prior to the 8-month visit, which led to a marked reduction in spirochaete counts for the rest of the study. The slope of the least squares linear regression line of mean % spirochaetes on time was computed for each subject. The subjects were ranked according to these slopes, and rank correlation was used to compare these rankings with the subjects' rankings on clinical improvement/deterioration (mean ΔY values). The rank correlation co-efficient was + 0.72 (p = 0.02).

Table 6.5 shows the test site means of % black-pigmented bacteroides over all seven visits. Very great variation was observed both between subjects and between visits within subjects, and no recognisable pattern emerged. The rank correlation between mean ΔY values and slope of linear regression line of mean % black-pigmented bacteroides on time was + 0.02.

Table 6.4 Test site mean % spirochaete counts from 0 - 12 months for individual subjects

Subject	Months							Rank by % spirochaete 'improvement'
	0	2	4	6	8	10	12	
A	6	10	12	12	1	1	1	2
B	2	3	6	11	5	5	1	5
C	5	10	9	7	3	4	10	3
D	15	14	13	16	12	5	3	1
E	4	3	1	4	4	4	2	4
F	20	24	25	20	30	30	38	11
G	3	4	1	3	3	4	3	7
H	8	7	6	6	4	7	10	8
J	11	14	18	6	17	15	16	9
K	12	12	15	17	14	13	12	6
L	4	6	7	7	9	15	12	10

Subjects ranked A - L in order of increasing tendency towards deterioration according to each individual's mean projected change in attachment level assessed by regression analysis.

Table 6.5 Test site mean % black pigmented bacteroides counts from 0 - 12 months for individual subjects

	Months							Rank by % BPB
Subject	0	2	4	6	8	10	12	'improvement'
A	0	4	14	8	0	1	5	4.5
B	8	9	1	4	16	18	4	8.5
C	2	5	3	0	3	2	0	3
D	52	10	1	15	40	6	37	2
E	1	1	3	3	1	1	5	6
F	*	6	27	12	26	42	3	10
G	3	4	3	4	15	7	4	8.5
H	1	1	3	13	2	12	10	11
J	4	0	2	1	4	4	5	7
K	2	13	15	5	2	4	6	1
L	1	25	7	34	25	3	8	4.5

Subjects ranked A - L in order of increasing tendency towards deterioration according to each individual's mean projected change in attachment level assessed by regression analysis.

* Missing data.

6.4 Discussion

The statistical analyses of the results of this study were designed to establish: (i) the effect of sampling at 2-monthly intervals on the subgingival microbiota; (ii) if there was a simple correlation between % spirochaetes or % black-pigmented *Bacteroides* species and the tendency towards deterioration or improvement of individual sites or subjects; and (iii) if these bacteriological variables could be used to predict attachment loss.

In the present study, the data were analysed using both the site and subject as the experimental units, the latter now being the favoured method (Blomqvist, 1985, Laster, 1985, Imrey, 1986).

No significant differences were observed in % spirochaetes between test sites (which were sampled at 2-month intervals) and control sites (which were sampled at the first and last visits only) when the data were analysed using either the site or subject as the experimental unit. This is consistent with the previous findings of Mousques *et al.* (1980) who reported that sampling caused only a transient decrease in spirochaete percentages, lasting only one week, and Magnusson *et al.* (1985) who described no systematic alterations in % spirochaetes when sampling was carried out on nine occasions over a 32-day period.

The possibility of a correlation between % spirochaetes and the occurrence of attachment changes was investigated. The mean % spirochaetes were calculated both on a site-specific basis and using the subject as the unit of statistical

analysis. Significant differences were observed only at the 12-month visit, when the % spirochaetes of both test and control sites were significantly lower in subjects who exhibited greatest gain in attachment. Since spirochaetes are not found in large proportions in shallow periodontitis-affected pockets (Rosenberg *et al.*, 1981, Evian *et al.*, 1982), it seems likely that the difference in spirochaete percentages observed was a function of reducing pocket depth, and not of attachment change *per se*. This explanation would also account for the trend towards slightly increased spirochaete counts with increasing tendency towards deterioration in both the 12-month data and the data accumulated over seven visits.

The lack of any significant difference between the *baseline* counts of deteriorating, stable and improving sites would indicate, not only that the presence of these organisms in individual sites cannot be used in a predictive capacity, but also that no association exists on a site-specific basis between spirochaetes and disease activity.

The lack of a simple correlation between the spirochaete proportions of improving, stable and deteriorating sites is reasonably consistent with the findings of other investigations. Claffey *et al.* (1985) investigated subgingival spirochaete counts at the end of a 1-year study of seven subjects, during which sites were classified as deteriorating or improving on the basis of linear regression analysis. When spirochaete percentages from the two categories of sites were compared, no statistically significant differences were found. Listgarten *et al.* (1984, 1986) found that spirochaete counts

in individual samples from deteriorated sites, and pooled samples from stable sites, were not significantly different. Slots *et al.* (1985) found that, while there was a significant association between spirochaetes and progressive loss of attachment, several active sites did not reveal these organisms. The only published data which support the predictive value of % spirochaete counts is by Listgarten & Levin (1981) and Listgarten *et al.* (1986), who demonstrated that disease-susceptible *subjects* had significantly higher baseline percentage spirochaete counts (using plaque, pooled from six sites per subject) than disease-resistant subjects. If more subjects had been investigated in the present study, it is conceivable that a trend may have emerged towards higher mean spirochaete counts in disease-susceptible subjects. However, we used our resources principally to observe the variations between a large number of diseased sites in a small number of individuals. What is clear is the need to interpret spirochaete counts, however derived, with considerable caution.

The results of the present study, as they relate to spirochaete populations, apply also in a similar fashion to black-pigmented *Bacteroides* species. That is, % counts of these organisms could not be used to identify or predict disease-active sites or subjects. Sampling, furthermore, had no effect on the proportion of black-pigmented bacteroides present.

In previous culture investigations of subgingival plaque, black-pigmented bacteroides isolates have been identified to species level, although data for only a few species (usually

B. gingivalis and *B. intermedius*) have been presented. Therefore, there is no information in the literature with which to compare the total black-pigmented bacteroides percentage counts which were obtained in the present investigation. It is generally accepted that no single culture medium will be equally effective in isolating all strains of a particular bacterial species, let alone all species of a particular genus. In addition, there are a number of factors which could result in variations in the isolation rate of the same bacterial species among different researchers using similar media, for example, the nature and concentration of blood and other growth factors added, and the manufacturer of the medium used. Therefore, the estimation of black-pigmented *Bacteroides* species using a single medium must inevitably be an approximation. The bacteroides isolates were not speciated in this study, since the intention of this study was to examine as many samples as possible using cheap and simple laboratory tests. However, we have isolated and identified *B. gingivalis* from subgingival plaque samples in other studies, using the same culture medium and techniques employed in this investigation.

The results of the present study revealed wide variation in the counts between individual sites with time, between different sites in the same individual, and between sites in different individuals (data not shown). However, none of the variation could be related to disease activity.

A close relationship between *B. gingivalis* and the presence of chronic periodontitis (Zambon *et al.*, 1985) or

disease-active periodontitis (Slots *et al.*, 1986) has been reported. Therefore, it is possible that, if the black-pigmented isolates from this study had been identified, a correlation between *B. gingivalis* and active sites may have resulted. On the other hand, the fact that Moore *et al.* (1983) failed to find a close relationship between either *B. gingivalis* or other black-pigmented *Bacteroides* species and chronic periodontitis could be interpreted as supporting the results of this study.

A number of factors may be responsible for the failure of this investigation to demonstrate a significant relationship between microbiological tests and the identification or prediction of disease-active sites. The laboratory techniques used for enumerating spirochaetes are different from the dark-field microscopy methods used by previous workers. However, there is no reason to believe that, if the conventional method had been used, a different result would have been obtained, since there is good evidence that the technique described by MacFarlane *et al.* (1986) gives reproducible results. Spiral platers have been used successfully by other workers to dilute and inoculate cultures in microbiological investigations of subgingival plaque (Loesche *et al.*, 1982), and again there is no reason to believe that the use of the more commonly used tube dilution method would have produced data which would have demonstrated a stronger association between % black-pigmented bacteroides and attachment changes.

Listgarten *et al.* (1984) offered a number of hypotheses to explain why *sites* with increased pocket depth did not

demonstrate significantly more spirochaetes than those derived from the six deepest probing sites in stable areas of the mouth: (i) an alteration in the host response without a change in the subgingival flora; (ii) a qualitative alteration in the plaque flora not detected by the laboratory techniques used; (iii) relatively brief episodes of disease activity, which may be accompanied by brief qualitative changes in plaque flora that cannot be detected by infrequent sampling. The same hypotheses may also explain the negative nature of our results. Furthermore, since the laboratory tests used are relatively crude, with inherent variables which are difficult to control accurately, it is unlikely that significant differences between samples from active and stable sites will be recorded consistently unless the differences are large. Since it seems likely that the microbial changes which are associated with the onset of active periodontitis are complex, it is perhaps not surprising that significant differences in microbiological variables were not demonstrated by the relatively crude techniques used both in this and other studies.

CHAPTER 7

CONCLUDING DISCUSSION

7.1 Introduction

This chapter continues the literature review which was begun in Chapter 1, and describes how knowledge of the occurrence, distribution, and progression of periodontitis has advanced since the present series of research projects was undertaken.

7.2 Cross-sectional radiographic studies — sampling methods

Since completion of the present cross-sectional study in 1982, very few full mouth radiographic surveys of marginal bone loss have been carried out in large representative populations. The paucity of such surveys can be attributed to cost, the logistics of using imaging techniques in field surveys and ethical considerations.

The present cross-sectional study overcame the logistical problem by using the facilities of a Dental Hospital Radiography Department, and the cost and ethical factors by using panoramic radiography rather than a full mouth series of intra-oral radiographs. By sampling the self-referred patient population of the hospital *before* examination and diagnosis had taken place, it was felt that the findings could be

extrapolated with caution to the general population.

Papapanou *et al.* (1988) obtained their sample of 531 sets of full mouth intra-oral radiographs by random selection from the records of the Department of Oral Radiology, University of Gothenburg, Sweden, but reported that, with respect to tooth loss and mean bone levels, the sample was similar to the general population of Scandinavia.

Salonen *et al.* (1991) reported the radiographic findings of an oral health survey of a randomly selected, age-stratified sample of 967 Swedish adults, yielding 732 dentate participants. Full mouth periapical radiographs were taken and a computer/digitiser recording method utilised to determine the amount of remaining bone support as percentages of root length. This choice of variable and the methods of data presentation make direct comparison with other surveys impossible, but, by inference, the pattern of periodontal destruction was broadly comparable to most of the other epidemiological studies to be discussed in this chapter.

In 1983, Hugoson, Laurell & Lundgren (1992) carried out a clinical and radiographic survey of a probability sample of 597 residents (550 dentate participants) of Jönköping, Sweden, evenly distributed into age levels of 20, 30, 40, 50, 60 and 70 years. Full mouth periapical radiographs were obtained and the subjects were grouped according to their general periodontal status. This study replicated one which had been carried out in Jönköping in 1973 (Hugoson & Jordan, 1982) — *see* Section 2.4.

7.3 Cross-sectional clinical studies — sampling methods

In recent years, the majority of epidemiological studies have adopted clinical attachment level measurements as the most appropriate method of assessing the destructive effects of periodontitis. Using a full mouth, circumferential probing approach, two population-based studies have provided valuable information on the distribution of attachment loss among adults of all ages in Kenya and Japan. Thus, Baelum *et al.* (1988) examined 1,131 persons, forming a stratified random sample of the entire population, aged 15—65 years, within a farming community with limited access to dental care in Kenya. In Japan, a detailed clinical examination was carried out on 319 citizens, 20—79 years-of-age, randomly selected from the population of Ushiku City. Findings were reported by Okamoto *et al.* (1988) and Yoneyama *et al.* (1988).

7.4 Comparison of findings from cross-sectional studies

Because these Swedish, Kenyan and Japanese studies, together with the present Scottish study, used relatively well standardised and objective measures of periodontal destruction (clinical attachment loss or proximal bone loss) to describe the distribution of periodontitis within large populations, valid comparisons of their findings can be made.

7.4.1 Generalized advanced periodontitis

Direct comparison of the present data with those of Papapanou *et al.* (1988) is not possible since the latter recorded bone loss in millimetres from a cemento-enamel junction reference point, rather than expressing bone levels as a percentage of optimum bone height. However, assuming an average root length of 15 mm (Papapanou *et al.*, 1988) and, therefore, an optimum bone height of 14 mm, the data presented by these investigators can be transformed from absolute values to percentages of optimum bone height (e.g., 6 mm bone level value ≡ 5 mm of bone loss against an optimum of 14 mm ≡ 35% of optimum bone height). Thus, in this Swedish study, bone loss *averaging* at least 35% of optimum bone height affected 2% of 40-year-olds, increasing to 18% of 65-year-olds. Similarly, in the present cross-sectional study, generalised bone loss averaging 25—50% of optimum bone height (Table 2.2) affected 3% of 35—39-year-olds increasing to 11.1% of 45—73-year-olds.

Evidence of the relatively low prevalence of *generalised* advanced bone loss had already been presented by Hugoson & Jordan (1982) in their first study of the Jönköping population in 1973 (*see* Section 2.4). Ten years later, in 1983, after a further sample of Jönköping residents had been obtained, it was shown that the proportion with *generalised* advanced bone loss (i.e. bone loss exceeding one third of root length) had increased among 40-year-olds from 2% to 4%, among 50-year-olds from 3% to 12%, among 60-year-olds from 8% to 32%, and among 70-year-olds from 6% to 42% (Hugoson *et al.*, 1992).

This *apparent* increase in experience of periodontitis in

cross-sectional surveys of the same target population, 10 years apart, may, perhaps, be attributed to the reduced level of edentulousness and increased number of teeth retained in 1983. Thus the lower prevalence figures obtained ten years earlier might represent a survival phenomenon, not evident to the same extent in 1983 when advanced periodontitis was not considered by the inhabitants or their dentists to be sufficient reason for extraction.

In Jönköping, between 1973 and 1983, the proportion of the 70-year-old sample, who were dentate, increased from 63% to 70%, and the average number of teeth retained by 70-year-olds increased from 13.3 teeth to 15.5 teeth. These findings suggest that tooth loss may be an important confounding factor in surveys of periodontal disease among elderly populations, raising the suspicion that periodontal disease experience could be higher than prevalence rates suggest.

In Scotland in 1978, when the present cross-sectional study was carried out, only 15% of over-65-year-olds were still dentate (Todd *et al.*, 1982), and, in the present study, the over-65-year-olds retained only 10 teeth, on average. These facts could explain why marginal bone loss among 60—70-year-olds in Jönköping in 1983 was so much greater than in 1973 or among 45—73-year-olds in Scotland in 1978, when the present study was carried out. The link between tooth retention and an increased prevalence of periodontal disease had already been noted by Douglass *et al.* (1983) in a comparison of national health surveys of the USA population carried out in 1960—62 and in 1971—74 using the PI.

7.4.2 Prevalence of advanced periodontitis

Although only a small proportion of individuals in the present cross-sectional study had *generalised* advanced destruction, the proportion of individuals with one or more sites affected by advanced destruction (greater than 50% of optimum bone height) was comparatively high, affecting only 1.5% of 20—24-year-olds, but increasing with increasing age to affect 29.4% of 40—44-year-olds and 50% of 45—73-year-olds (Table 2.3).

Similar data are reported by Okamoto *et al.* (1988) from their Japanese study. They found that attachment loss of 7 mm or more (i.e. ≥ 50% of root length) affected 4% of 20—29-year-olds, 6% of 30—39-year-olds, 25% of 40—49-year-olds, 31% of 50—59-year-olds and 55% of 60—79-year-olds. Whether the somewhat higher prevalences in the present study, compared to the Japanese study, represent real differences between the populations, or errors in data transformation, is not important. Both series of data emphasise the common occurrence in middle-aged people of advanced periodontitis affecting at least one tooth.

7.4.3 Distribution of teeth affected by advanced periodontitis

In spite of the fact that advanced periodontitis is widespread in the population, only a small proportion of teeth seem to become affected by advanced disease. In the Japanese study, for example, Yoneyama *et al.* (1988) reported that only approximately 5% of proximal surfaces in 60—69-year-olds were affected by loss of attachment of 7 mm or more. Baelum *et al.* (1988) reported that 8% of tooth surfaces in 55—65-year-old

Kenyans had attachment loss of 7 mm or more. Similarly, in the present study, only 8.4% of teeth in 45—73-year-olds had proximal bone loss amounting to more than 50% of optimum height (Table 2.4). Furthermore, when the present study population was compared with the Kenyan sample (Baelum *et al.*, 1988), a Swedish population (Papapanou *et al.*, 1988) and a small population of Tanzanians (Baelum *et al.*, 1986), the distribution of tooth surfaces affected by advanced disease in each population was found to be similar (Figure 3.1).

One further study should be mentioned:- in fact, the largest of its kind yet to be reported — a cross-sectional study of the United States employed population, which was carried out by the National Institute of Dental Research in 1985-86, and is described by Brown, Oliver & Löe (1990). A probability sample of 15,132 adults, 18—64 years-of-age was surveyed and a half-mouth design employed by randomly selecting one maxillary quadrant and one mandibular quadrant, and examining the mid-buccal and mesio-buccal sites on each tooth. However, the validity of this partial recording system has been much criticised: several investigators have demonstrated that it substantially underestimates the prevalence of advanced disease (for review, *see*: Douglass & Fox, 1993 ; Papapanou, 1994).

7.4.4 Early-onset advanced periodontitis

It is now customary to recognise two principle forms of periodontitis: 'chronic adult' and 'early-onset' periodontitis. The former is by far the more prevalent, and only a small

minority of individuals are thought to suffer from early-onset disease. In adolescents, early-onset advanced periodontitis is known as juvenile periodontitis. The commonest form is localised juvenile periodontitis which is characterised by severe attachment loss, principally affecting incisors and first molars. No cases of juvenile periodontitis were identified among the 155 16—19-year-old subjects in the present study. In this age-group, only one tooth (a second molar) was found with bone loss greater than 50% of normal bone height. The absence of juvenile periodontitis from this study is not surprising since its prevalence in a United Kingdom adolescent population is reported to be only 0.1% overall, and only 0.02% among Caucasians (Saxby 1987).

In young adults, 20—35-years-old, early-onset advanced disease, affecting most teeth, is sometimes known as rapidly progressive periodontitis (World Workshop in Clinical Periodontics, 1989). Rapidly progressive periodontitis, however, is not a well defined disease entity and no consensus has been reached as to the precise criteria for such a diagnosis. Marginal bone loss amounting to one third of root length has been chosen arbitrarily by previous investigators (Hørmand & Frandsen, 1979) as the threshold for advanced bone loss. Applying this criterion, Hugoson *et al.* (1992) found only three individuals among 100 20-year-olds and 100 30-year-olds with that amount of *generalised* destruction, giving a prevalence rate of 1.5%. Likewise, Papapanou *et al.* (1988), in their radiographic study, found only one individual out of 152 young adults, 20—35 years-old, with a mean bone level

value as much as 6 mm (one third of root length). In the present study, only two (0.7%) individuals out of 302 20—34-year-olds exhibited mean bone loss scores amounting to more than 25% of optimum bone height (Table 2.2). There is, therefore, good agreement between these three studies that rapidly progressive periodontitis, if it does indeed exist as a true entity, affects only a small fraction of the young adult population.

7.4.5 Prevalence of early periodontitis

Although severe periodontal destruction is uncommon among children and young adults, it is well established that minor amounts of attachment loss are highly prevalent. Clerehugh, Lennon & Worthington (1990) carried out a 5-year longitudinal study of 167 British adolescents initially 14 years-of-age, from a low socio-economic area. This was not intended as a representative sample of the population, but a group which were expected to develop periodontitis. They determined the prevalence of attachment loss by examining 12 surfaces in each mouth: the mesial surfaces of first molars, first premolars and central incisors were examined for evidence of a reduction in attachment level of 1 mm or more. The prevalence increased from 37% of subjects at 16 years to 77% at 19 years, and the site prevalence increased from 7% at 16 years to 31% at 19 years.

In the present study, in which all proximal surfaces were scored, except for those which were radiographically unmeasurable, 84.5% of subjects (Table 2.3) and 18.3% of teeth

(Table 2.4) in the 16—19-year age-group had evidence of marginal bone loss. Therefore, although the present study used a relatively crude measurement technique, there is, nonetheless, quite good agreement with the data of Clerehugh *et al.* (1990) which was obtained by a very precise detection method, albeit at a limited number of sites. Of course, prevalence estimates of periodontitis are a function of the criteria used for diagnosis, and lower rates are obtained when assignment of a diagnosis of periodontitis depends on attachment level reductions of > 2 mm instead of ≥ 1 mm. Thus, Källestål, Matsson & Holm (1990) reported that, in a full mouth study of periodontal conditions in 283 18-year-olds from Northern Sweden, only 4.9% exhibited proximal attachment loss greater than 2 mm at one or more sites. This is reasonably in line with the further finding of Clerehugh *et al.* (1990) that 14% of 19-year-olds had attachment loss of 2 mm, but none exhibited attachment loss greater than 2 mm.

7.4.6 Highly susceptible populations

The PI (Russell, 1956), which combines gingivitis and loss of attachment in a single score, has, since the late 1970s, been progressively replaced by disaggregated indices to record the destructive effects of periodontitis. P.I. scores were probably unduly influenced by high levels of plaque and gingivitis. This would help to explain why pre-1982 literature (*see* Chapter 1) revealed considerable variation in the reported prevalence and severity of periodontal disease throughout the world. In contrast, this review of more recent literature

suggests relatively good agreement between populations in the reported occurrence and distribution of the destructive effects of periodontitis.

Nevertheless, the populations of two South Pacific Islands, Tonga and West Samoa (Cutress *et al.*, 1982), and a population of Sri Lankan tea labourers (Löe *et al.*, 1978; 1986) have been identified with levels of attachment loss far beyond the 'norm' for other countries (Baelum, Manji & Fejerskov, 1991). The extent to which these findings can be attributed to the very high plaque levels within these communities is not known. The Kenyan population described by Baelum *et al.* (1988) also had very poor oral hygiene, but much less periodontitis. Furthermore, the amount of periodontitis in West Samoa, although considerable, was significantly less than in Tonga, a finding which could not be attributed to differences in oral hygiene, since equal levels of plaque, calculus and gingivitis were found in both populations by the same examiners using the same diagnostic criteria. Since these two populations were ethnically similar and shared similar diets, lifestyle and oral hygiene practices, it is tempting to conclude that the difference in disease may reflect a greater genetic predisposition to periodontitis among Tongans; each island being a relatively closed community with a restricted gene pool.

It is also interesting to note that, both among the Sri Lankans and the South Pacific islanders, although overall periodontal conditions were very poor, the disease was unevenly distributed within these populations, and only a small

proportion of individuals appeared to be suffering multiple tooth loss due to periodontal disease. Thus, the existence of highly susceptible *groups* appears to be a characteristic of most, if not all, populations regardless of their overall experience of periodontitis.

7.5 Caries as a risk indicator for periodontitis

The present cross-sectional study has shown, in common with established belief, that age, sex and socio-economic status are determinants of periodontitis. However, such great variation existed within these subgroups that none of these variables could be considered to be reliable indicators of the presence of periodontitis or predictors of its progress. The study was, therefore, extended to include a radiographic assessment of carious and restored teeth among the 800 subjects, so that the susceptibility to caries and periodontitis within the same individual could be compared. Since caries and periodontal disease share a common aetiological factor of major importance — dental bacterial plaque — and since caries is a disease which occurs earlier in the life-time of the individual, it could be postulated that a high caries experience might be used to predict periodontitis. No association between caries and periodontitis was found, however, suggesting that the major risk factors for caries and periodontitis are quite different.

Frentzen, Schüler & Nolden (1990) reached a similar conclusion from a cross-sectional study of caries and

periodontal disease in 2,200 dental patients aged between 18 and 80 years in Germany. Using the Community Periodontal Index of Treatment Needs (Ainamo *et al.*, 1982), scores were obtained for each sextant and group mean values were compared with group DMFS scores. No correlation could be established between caries and periodontal treatment needs.

7.6 Other risk indicators for periodontitis, identified by cross-sectional study

Recently, Grossi *et al.* (1994, 1995) have reported the findings of a cross-sectional clinical and radiographic survey of 1426 subjects, 25—74 years-old, evenly distributed in five age categories. The subjects were selected from various sources to display a broad variation in dental explanatory variables and wide range of periodontal disease experience. It was shown that age, smoking, diabetes mellitus and the presence of subgingival *Porphyromonas gingivalis* and *Bacteroides forsythus* were significant risk indicators for clinical attachment loss and marginal bone loss. These associations remained valid after controlling for race, gender, socioeconomic status, income, education and oral hygiene.

7.7 Longitudinal studies

The present cross-sectional study, and numerous others during

the last 10 years, have confirmed that periodontitis is highly prevalent both at a subject and at a site level, but these studies have also demonstrated a much lower prevalence of generalised severe periodontitis than was formerly suggested. Most lesions, by inference, progress too slowly to threaten tooth survival. Thus, with the recognition that there exists a susceptible minority of teeth and individuals, periodontal research has become focused on what characteristics are peculiar to the highly susceptible or 'high risk' group, and how such teeth and individuals may be identified in time for preventive action to be taken.

Most of the recent research in this field has involved cohort studies, where changes in attachment level over a specified period of time are measured, and attempts are made to relate this destructive activity to various clinical or laboratory findings, which could then serve as markers of disease susceptibility.

7.7.1 Reliability and validity of attachment level measurements

A major weakness of the early investigations, including the present longitudinal study, was the low incidence of progressing disease and large measurement error (relative to disease magnitude) associated with attachment loss measurement. Measurement error may be attributed to variations in probe positioning (Watts, 1989) and angulation (Karim, Birek & McCulloch, 1990), probing pressure (Van der Velden, 1979), and the practice of 'rounding' readings to the nearest millimetre.

Because of these factors, the standard deviation of replicate measurements has been assessed as 0.8 mm within a range of 0.5—1.3 mm (Haffajee *et al.*, 1983*b*).

There are many ways to overcome errors associated with probing reproducibility. Duplicate or triplicate measurements may be made at each time-point, and an average value calculated. Sequential measurements over time may be analysed by the regression, tolerance, running medians or cumulative-sum methods (Goodson, 1992). Arbitrary cut-off levels may be chosen to identify deteriorating sites, balancing the probability of type 1 and type 2 statistical errors. Alternatively, individual site and patient thresholds may be used, based on the standard deviations of replicate measurements according to tooth type, site location and pocket depth.

Recently, probing techniques have been refined with the introduction of electronic pressure-sensitive probes, which provide controlled force application, automated measurement and computerized data capture. Automated probe measurements are resolved to the nearest 0.1 mm or 0.5 mm, depending on the instrument, compared to 0.5 mm or 1.0 mm for the manual probe, and a more continuous distribution of measurements is obtained. On the other hand, automated probes have poorer handling characteristics and probe tip positioning may be difficult.

There is limited evidence of improved reproducibility when automated probes are used to detect attachment levels. Some studies have demonstrated similar levels of reproducibility for conventional and automated probing, while one group of

investigators has reported that better reproducibility was achieved with the conventional probe (Wang *et al.*, 1995). On the other hand, using the prototype of a technologically advanced, automated probe, Jeffcoat & Reddy (1991) showed that the average standard deviation of difference between repeated measurements was only 0.2 mm, which compares with 0.8 mm for manual probing (Haffajee *et al.*, 1983*b*).

Using an automated probe in a longitudinal study, Jeffcoat & Reddy (1991) showed that small increments of disease activity could be detected, thereby giving a higher incidence of attachment loss than that usually determined by conventional probing. They observed attachment loss at 29% of sites, probed at 2-month intervals over a 6-month period, using the cumulative-sum method of analysis with a 0.4 mm threshold for destructive change. Furthermore, linear regression analysis of the data revealed that 76% of active sites exhibited a linear pattern of attachment loss, supporting the findings of an earlier study by Badersten *et al.* (1985). In the latter 2-year post-therapy study of 33 patients, during which no subgingival instrumentation was performed, 73% of deteriorating sites exhibited a linear pattern of gradual attachment loss, based on nine sequential measurements with a manual probe.

These observations — that most attachment loss may occur in a gradual continuous manner, rather than by acute bursts of activity — seem to justify the choice of linear regression analysis as the analytical method for the present longitudinal study. Although linear regression analysis is insensitive to abrupt changes in attachment level, it is very sensitive to

continuous changes (Haffajee *et al.*, 1983*b*). However, even if attachment loss proceeds most often at a gradual, continuous rate, other patterns of disease activity may occur, involving loss of attachment of variable intensity and duration with variable periods of remission or repair. Yang *et al.* (1992) performed replicate attachment level measurements at monthly intervals for one year using an automated probe at 1061 sites in untreated periodontal patients. These authors found that no single method of attachment loss could fully explain the variation in data, and that, in the course of one year, attachment level change at any given site may not follow the same model. Therefore, a further important concern in the longitudinal analysis of attachment levels is the possibility of imposing an inappropriate mathematical model upon the underlying biological events.

Not surprisingly, the reported incidence of attachment loss varies according to the method of analysis (Haffajee *et al.*, 1983*b*), the chosen threshold value for attachment loss (Goodson, 1992) and the population studied, whether a probability sample of a large community with a low annual rate of attachment loss of 0.35% of sites per year (Lindhe *et al.*, 1989), or a population of untreated periodontal patients with a higher annual rate of 1.9% of sites per year (Lindhe *et al.*, 1983). In the present longitudinal study (*see* Section 5.3.1), incidence values for attachment loss were: 1.9% of sites per year when an observed loss of $\geq$ 3 mm was chosen as the threshold; 9.8% of sites per year for a $\geq$ 2 mm attachment loss threshold; and 12% of sites per year, employing regression analysis with ΔY of $\geq$ 1 mm and $p < 0.05$.

It is disturbing, but not surprising to note that there is poor agreement between different statistical methods of determining attachment loss (Haffajee *et al.*, 1983*b*), and that probing errors may account for 25—50% of sites identified with attachment loss (Halzonetis, Haffajee & Socransky, 1989; Lindhe *et al.*, 1989).

Finally, changes over time in the inflammatory status of the tissues can result in errors of interpretation; increased probe penetration, due to increased inflammation, being wrongly interpreted as irreversible connective tissue attachment loss (Listgarten, 1980).

Thus, whatever detection methods are employed for investigations of progressive periodontal breakdown, the findings of such studies should be interpreted with caution because of the problems of reliability and validity, outlined above.

7.7.2 Clinical parameters as predictors of disease activity

The testing of clinical parameters as markers or predictors of disease activity in untreated subjects has continued in recent years.

Löe *et al.* (1986), in their longitudinal study of Sri Lankan tea labourers, identified three subpopulations based on proximal attachment loss and tooth mortality rates. At 35 years-of-age, mean loss of attachment in the three groups amounted to 9 mm, 4 mm and 1 mm respectively. Yet, all three groups at all age levels exhibited similar amounts of gross plaque accumulation and severe gingivitis.

Halzonetis *et al*. (1989) carried out a study, similar in scope to the present longitudinal study, involving eight untreated subjects monitored every 2 months for one year. Duplicate attachment level measurements were carried out. Data were analysed at the site level, 5.4% of sites showing evidence of attachment loss. Severe disease at baseline, as expressed by probing depths and attachment levels, showed the strongest relationship to disease activity.

In the present longitudinal study, *relative* rather than *absolute* attachment levels were measured, and there was no correlation between baseline probing depth and subsequent attachment loss. The present investigation, however, excluded sites with probing depths less than 4 mm, whereas Halzonetis *et al*. (1989), and many other investigators, have counted all tooth surfaces, including those with shallow probing depths and no evidence of disease at baseline.

Lindhe *et al*. (1989) and Haffajee *et al*. (1991) evaluated the relationship between various baseline clinical variables and subsequent attachment loss over a 24-month period in a population of 271 untreated Japanese citizens. A diagnosis of disease progression was made when an observed loss of attachment of 3 mm or more occurred. After 12 months, 27% of subjects exhibited attachment loss at one or more sites, and these subjects had greater disease severity at baseline, as expressed by the proportion of sites with severe attachment loss or deep pockets (Haffajee *et al*. 1991). After 24 months, a total of 0.7% of sites and 40% of subjects were affected by attachment loss (Lindhe *et al*., 1989). Progressive destruction

was most often detected in older subjects, at molars, at proximal surfaces and at surfaces with initially advanced loss of attachment or deep pockets (Lindhe *et al.*, 1989). The predictive value of these observations, however, must be assessed within the context of a high estimated probing error and low site incidence of attachment loss in this study.

Although some longitudinal studies appear to show a relationship between pre-existing destruction and subsequent disease activity, or between intraoral location and subsequent disease activity, it can be argued that higher thresholds of attachment loss should be applied at these sites. This is because the reliability of probing measurements is poorer at deep pockets and proximal sites (Badersten, Nilvéus & Egelberg, 1984), and at molar sites and sites of severe attachment loss (Loos, Kiger & Egelberg, 1987). Nevertheless, longitudinal investigations of proximal radiographic bone height, using standardized techniques with relatively *good* reproducibility, among individuals not receiving systematic periodontal treatment, support the idea that the risk of progressive destruction increases with increasing severity of pre-existing destruction (Bolin, Lavstedt & Henrikson, 1986; Lavstedt, Bolin & Henrikson, 1986; Albandar, 1990). Furthermore, Machtei *et al.* (1993), using an automated probing system and adopting a multiple threshold approach to correct for factors which contribute to the error of attachment level measurements, found that the frequency and magnitude of clinical attachment loss increased substantially with increasing baseline probing depth. These authors also observed that, although the frequency of

attachment loss at molar sites was no different from non-molar sites, the magnitude of attachment loss at *active* molar sites was substantially greater than at *active* non-molar sites.

Most longitudinal studies of attachment loss have been undertaken in adult subjects, often with significant pre-existing periodontal disease and presumably affected by ongoing destruction. In adolescents, although there is a greatly reduced risk of incurring large amounts of periodontal breakdown, the absence of deep pockets at baseline enables progressive attachment loss to be detected reliably at mesio-buccal surfaces in increments of 1 mm (Clerehugh & Lennon, 1986). Thus, Clerehugh *et al.* (1990), in their 5-year longitudinal study of 167 British teenagers (*see* section 7.4.5), observed that those who developed loss of attachment ≥ 1 mm in the first two years of the study still had a higher mean, extent and severity of loss of attachment after the following three years. Disease progression was detected much more frequently at maxillary first molars and mandibular central incisors than first premolars and maxillary central incisors, and there was a significant correlation at the subject level between the presence of subgingival calculus at baseline and the subsequent development of attachment loss. Progressing sites, furthermore, had significantly more plaque, subgingival calculus and gingival inflammation than non-progressing sites at the baseline examination and throughout the study (Clerehugh *et al.*, 1995). The predictive value of plaque and gingivitis for subsequent attachment loss, observed in this study of the earliest stages of periodontitis, may not,

of course, apply in the later stages, when destructive changes at the base of a deep pocket, and an inflammatory reaction in the marginal gingival tissues, may occur independently.

Many other longitudinal investigations, of up to 28 years duration (Ismail *et al.*, 1990), have been carried out, involving two or more sequential measurements of attachment level or bone level in populations with unrestricted access to periodontal care. Egelberg & Claffey (1994), reviewing studies both of treated and untreated subjects, conclude that there is an increased risk of further deterioration in sites and subjects with evidence of pre-existing loss of periodontal support or deep pockets. They point out that this is not surprising since past susceptibility is likely to continue into the future. They note, furthermore, that the risk of additional attachment loss increases with increasing age, and is greater at molars and proximal surfaces, and, although plaque and gingival redness scores are poorly correlated with subsequent attachment loss, repeated bleeding on probing from deep pockets at sequential examinations has modest predictive power. It has also been shown that angular bone defects are at greater risk of additional bone loss than sites with horizontal bone loss (Papapanou & Wennström, 1991).

It must be stressed that the predictive powers of these clinical and radiographic examination findings are limited and that predictions may be more valid for subjects than for specific sites because of the interdependence of sites within subjects.

7.7.3 Bacteria as predictors of disease activity

In recent years, numerous studies have been performed to establish which bacteria are aetiologically associated with periodontitis. Bacterial pathogens among the subgingival flora of periodontitis have been identified by several means: from cross-sectional association studies in which they have been more frequently detected, and at higher levels, in cases than in healthy controls; from antibody response studies; by examining virulence properties of oral organisms; and from longitudinal studies.

From a review of the literature, Moore & Moore (1994) listed 35 species of candidate pathogens among which *Fusobacterium nucleatum* was singled out as the principle and most frequent isolate at all stages of gingivitis and periodontitis. Haffajee & Socransky (1994), on the other hand, consider that *Actinobacillus actinomycetemcomitans* and *Porphyromonas (Bacteroides) gingivalis* are the bacteria with the strongest relationship to periodontitis.

7.7.4 Retrospective studies of the subgingival flora

The great majority of longitudinal investigations have been retrospective case-control studies, which have followed sites clinically over time, but which have characterised specific bacteria from these sites only *after* attachment loss has occurred. The main reason for this retrospective approach has been the prohibitive cost of cultivating the subgingival flora from a large number of patients and sites at baseline in the expectation that only a small subset would show disease progression.

Using this retrospective approach, Dzink, Socransky & Haffajee (1988) and Moore *et al.* (1991) detected a wide range of subgingival species, but found no significant differences in the flora of active and inactive sites, if the differences were corrected for multiple comparisons. On the other hand, Bragd *et al.* (1987) and Mandell, Ebersole & Socransky (1987), have demonstrated, also by retrospective analysis, a strong association between specific bacteria and disease progression.

7.7.5 Prospective studies of the subgingival flora

It is well recognised that deep pockets or sites of recent attachment loss may contain, not the causative organisms, but those whose growth have been stimulated by blood and serum from recent tissue destruction. The identification of bacteria as risk factors or predictors, therefore, requires prospective studies which monitor sites on a longitudinal basis both clinically *and* microbiologically, so that microbiological data are available before destruction has taken place.

While the present longitudinal study is one of the few which have obtained sequential microbiological data both before and after periodontal destruction, and throughout the clinical monitoring period, it was not considered practicable, because of cost factors, to speciate the black-pigmented bacteroides. Other investigators have sought to contain the costs of their projects by: monitoring a limited number of sites; storing plaque samples under liquid nitrogen and, later, selecting which ones to culture; collecting bacteriological samples only at baseline; or isolating a limited range of bacteria.

Wennström *et al.* (1987) carried out a prospective study, the first of its kind, monitoring 44 deep pocket sites in 30 patients. At baseline, 57% of sites contained either *A. actinomycetemcomitans*, *P. gingivalis* or ≥ 5% *Prevotella (Bacteroides) intermedia*, but only 20% of these infected sites showed loss of attachment ≥ 2 mm during the following year. No attachment loss occurred at the sites which did not harbour *A. actinomycetemcomitans*, *P. gingivalis* or ≥ 5% *P. intermedia*, leading the authors to conclude, that absence of these 'indicator' bacteria was a better predictor of no further loss of attachment, than was the presence of these bacteria for disease progression.

Ashley, Gallagher & Wilson (1989) obtained microbiological samples, which they stored under liquid nitrogen, from mesial and distal surfaces of all first molars in 89 adolescents, initially 14 years-old, at 6-month intervals for 2 years. Attachment levels were monitored during this period. Their conclusions were limited by the low incidence of attachment loss, which affected only six sites. However, at the 12-month visit, 6—12 months prior to the diagnosis of attachment loss, black-pigmented bacteroides were elevated in pooled samples from all eight sites of each affected subject. There was, however, no significant difference in baseline counts of *A. actinomycetemcomitans*, *P. gingivalis* or *P. intermedia* between active and control sites.

In a 3-year prospective study of 98 patients on regular maintenance, previously treated for periodontitis, Listgarten *et al.* (1991) found that baseline screening for *A.*

actinomycetemcomitans, *P. gingivalis* and *P. intermedia* did not reliably predict future episodes of disease.

Skaar *et al.* (1992) followed 16 subjects for up to 56 months and showed that teeth with deep pockets were more likely to be lost when these pockets contained *A. actinomycetemcomitans* at baseline.

7.7.6 Prospective studies with new diagnostic techniques

With the recent introduction of immunological and DNA probe techniques, rapid species identification and more rigorous testing of causality can now take place in prospective studies of periodontal disease aetiology.

Thus, using DNA probes, Socransky & Haffajee (1992) enumerated suspected pathogens at 2-month intervals in 67 subjects with prior evidence of periodontal destruction, and found that a number of microbial species were related to the risk of further attachment loss.

Using radioimmunoassay techniques, and controlling for various clinical parameters, Wolff, Dahlén & Aeppli (1994) obtained a positive correlation between the baseline presence of *P. gingivalis* (without *A. actinomycetemcomitans*) in patients with early periodontitis and the proportion of individuals experiencing attachment loss during the subsequent 58 months. In this study of patients with early periodontitis, disease activity could not be correlated to baseline presence of *A. actinomycetemcomitans*.

7.7.7 The value of microbiological diagnoses

It is likely that clinical monitoring of attachment levels remains a source of variability that could contribute significantly to the relatively poor correlation between microbiological and clinical data, and it is clear that, even using modern techniques of species identification, reliable microbial indicators of impending periodontal breakdown have not been found.

Socransky & Haffajee (1992) have suggested that, even if microbiological analysis correctly identifies the pathogenic species present, the following conditions must be met for disease to occur: the pathogen(s) must be of a virulent clonal type; the pathogen(s) must be present in sufficient numbers; the host must be susceptible to the pathogen(s); other bacterial species, which might inhibit the disease process, must be absent; and the local environment must favour expression of the organism's virulence properties. It is, therefore, likely that better predictions of progressive disease could be made by combining clinical, microbial, host and environmental markers.

7.8 Concluding Remarks

This Chapter has comprised a compilation of recent studies on the occurrence, distribution and progression of periodontitis which support, extend and, in some cases, challenge the findings of this author's own investigations. Many issues have

yet to be fully resolved, but sufficient is known for a tentative model of periodontitis to be constructed.

The prevalence, severity and extent of periodontitis is greater in males than in females, and increases with increasing age. With a few known exceptions, it appears to affect ethnically and geographically different populations to a similar degree. Although some studies have shown that periodontitis is more severe in certain racial groups, it is not clear whether this association is attributable to some intrinsic effect of race, or is a function of confounding factors. Smoking, for example, is now well established as a major risk factor for periodontitis, and could be a confounding factor in cross-sectional studies which seek to link periodontitis with demographic variables. Thus, the existence of increased levels of periodontitis in lower socio-economic groups, while previously attributed to their higher plaque levels, may also be related to smoking habits.

Periodontitis is common in adolescence, and almost universal by early adulthood, by which time a high percentage of teeth are affected. Destructive changes due to periodontitis, however, rarely exceed 1—2 mm of attachment loss at any site.

Severe periodontitis in adolescence is usually manifested as localised juvenile periodontitis. According to one study, localised juvenile periodontitis affected 0.1% of British adolescents. Severe periodontitis involving *many* teeth probably affects fewer than 2% of young adults.

In middle-age, although severe periodontitis is quite

prevalent, *generalised* severe periodontitis affects only about 5% of the population. On the other hand, the current trend towards reduced edentulousness and tooth retention in old age has revealed a considerable potential for advanced periodontal disease among the elderly.

Cross-sectional studies have identified 'high risk' or highly susceptible populations and population subgroups, and susceptibility may also vary greatly within individuals.

Periodontitis may progress at a continuous rate, either slowly or rapidly. Alternatively, progression may be episodic, acute episodes being interspersed with periods of remission or repair. Different patterns of progression may affect the same site at different times, and prolonged remission may not be uncommon.

There are no reliable predictors of progressive destruction. Although a number of risk indicators have emerged from cross-sectional and longitudinal studies, none possess the necessary criteria of sensitivity and specificity to be regarded as powerful prognostic tools. Lack of good access for plaque control would help to explain why molars and proximal surfaces are most at risk from periodontitis, and ongoing susceptibility would explain why pre-existing deep pockets, sites of severe attachment loss and angular bone defects are most at risk from progressive destruction.

There is no doubt that periodontitis is caused by microorganisms. Longitudinal studies have shown that, in its early stages, periodontitis is positively associated with pre-existing supragingival plaque, gingivitis and subgingival

calculus. However, the absence of major differences in prevalence and extent of *advanced* periodontitis between populations with different standards of oral hygiene suggests that, once periodontitis is initiated, progressive destruction is caused by factors other than supragingival plaque.

It seems likely that, once subgingival plaque is established, its dependence on supragingival plaque accumulation is substantially diminished, and further disease progression is dependent on the composition of the *subgingival* flora and its interaction with host inflammatory and immune mechanisms.

Although a wide range of subgingival pathogens has been found, no organism or group of organisms has been identified which is consistently associated with advanced or progressing periodontitis. This would suggest that host susceptibility could be a factor of considerable importance in determining whether certain individuals or specific sites experience progressing periodontitis once it has become established.

A similar case-mix of periodontitis-resistant and periodontitis-susceptible individuals might be expected in most populations, in common with many other diseases of bacterial origin. This would help to explain why a similar distribution of *advanced* periodontitis is common to many parts of the world, in spite of considerable differences in living conditions, oral hygiene practices and availability of dental care. Only small amounts of plaque would be required to initiate periodontitis in a susceptible subject, so that the population frequency of *advanced* disease would not be closely related to the general

standard of oral hygiene within the population. This does not mean that plaque control is not the mainstay of prevention and treatment for periodontitis at the present time; but, for those with the greatest susceptibility, standards of plaque control must be very high to prevent progressive disease, or recurrent breakdown after treatment.

APPENDIX

Publications:-

Jenkins, W.M.M. & Mason, W.N. (1984) Periodontitis in the United Kingdom: A literature review. *British Dental Journal*, **156**, 43—45.

Jenkins, W.M.M. & Mason, W.N. (1984) Radiographic assessment of periodontitis: A study of 800 unreferred patients. *British Dental Journal*, **156**, 170—174.

Jenkins, W.M.M. & Kinane, D.F. (1989) The 'high risk' group in periodontitis. *British Dental Journal*, **167**, 168—171.

Kinane, D.F., Jenkins, W.M.M., Adonogianaki, E. & Murray, G.D. (1991) Cross-sectional assessment of caries and periodontitis risk within the same subject. *Community Dentistry and Oral Epidemiology*, **19**, 78—81.

Jenkins, W.M.M., MacFarlane, T.W. & Gilmour, W.H. (1988) Longitudinal study of untreated periodontitis: (I). Clinical findings. *Journal of Clinical Periodontology*, **15**, 324—330.

MacFarlane, T.W., Jenkins, W.M.M., Gilmour, W.H., McCourtie, J. & McKenzie, D. (1988) Longitudinal study of untreated periodontitis: (II). Microbiological findings. *Journal of Clinical Periodontology*, **15**, 331—337.

Jenkins, W.M.M. & Allan, C.J. (1994) Classification and Epidemiology. In *Guide to Periodontics* (3rd edn), Ch 5, pp 41—50. Oxford: Wright.

Periodontitis in the United Kingdom

A Literature Review

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During the last 15 years a number of clinical studies have been published on periodontal disease experience in the United Kingdom. The conflict in these reports seems to reflect the lack of reliable diagnostic criteria.

Dental manpower, both graduate and ancillary, is currently under review, while a greater proportion of health care resources is being earmarked for dental health education.[1] However, resources cannot be properly allocated without a fundamental appreciation of the occurrence and distribution of dental and periodontal disease. The epidemiology of caries is comparatively well documented and the reported decline of caries prevalence in developed countries throughout the world[2] suggests that periodontal disease will be the major preoccupation of dental personnel in the foreseeable future. It is important, therefore, to make sure that our epidemiological data on periodontal disease are both accurate and informative.

There are abundant data on the prevalence and severity of chronic gingivitis in children and adults. From a public health standpoint, however, it is the destructive consequence of periodontal disease that is most relevant and this cannot be assessed from gingivitis statistics. It is well recognised that destructive periodontal disease (chronic periodontitis) represents the apical extension of an inflammatory lesion which is initiated in the gingiva. Nevertheless, gingivitis lesions do not necessarily progress to periodontitis[3] and the rate of progress of destructive lesions varies greatly at different gingival sites in one mouth and between different individuals.[4] Collecting data on the occurrence and distribution of chronic gingivitis, therefore, can give little indication of the threat to tooth survival posed by chronic periodontitis.

To obtain a profile of chronic periodontitis within a population that would be helpful for determining treatment priorities the following must be ascertained: the number of individuals affected; the number of teeth affected; a frequency distribution of individuals according to severity of the disease.

Periodontitis: United Kingdom data

A number of dental surveys, reporting the prevalence and severity of chronic periodontitis, have already been conducted in the United Kingdom. These include the national surveys of adult dental health in England and Wales in 1968,[5] in Scotland in 1972[6] and in the United Kingdom as a whole in 1978.[7] Periodontal data have also been obtained independently from studies in Wales,[8] England[9] and Northern Ireland.[10] These national and independent surveys are distinguished by marked differences in methodology.

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For each national survey, subjects were selected from the electoral register, with a supplementary sample of young persons, to give a total sample which was as representative as possible of the population in the age range 16 years and over. In order to complete each survey within a reasonable time, it was necessary to recruit a large number of examiners since the sample was dispersed over the length and breadth of the country. Not all examiners were experienced in the examination of periodontal disease and, although each survey was preceded by a period of intensive training, it was necessary to use rather crude indices of disease status in an attempt to reduce individual examiner variation to an acceptable level. Furthermore, to achieve a high response rate the dental examinations were conducted in the subjects' homes, thus limiting the scope of the examination. It was acknowledged that these constraints would underestimate true disease levels.

While the national surveys involved samples that were broadly representative of the populations of each country, the independent studies were confined to part of the population of a certain district,[8] or to factory populations.[9,10] Swallow and Adams[8] set out to examine all adults within two defined areas in the Rhondda Valley of South Wales. Sheiham[9] obtained his sample by random selection from the lists of employees of industrial units, two in London and one in Warrington. Sheiham and Dimmer's study of the Northern Ireland population[10] was carried out on a random sample of white and blue collar workers at a large Belfast factory. Despite the lack of similarity among these populations and those of the national surveys, one would not anticipate that these differences could be large enough to account for wide variations in periodontal disease.[9] Any marked difference that might emerge between the results of the national and independent surveys is more likely to be attributable to differences in the conduct of the examinations. The independent surveys were carried out by one[9,10] or two[8] examiners with the subject seated in a dental chair, using well established criteria of periodontal disease rather than criteria specifically designed for a large number of examiners with different backgrounds working under less than ideal conditions. On the other hand, a single-examiner or even a two-examiner system carries with it the risk that the results may reflect the individual prejudices of the examiners.

Because of the variations in survey methodology and in the compilation of data, these studies do not all yield the same type of information. Nevertheless, in this review an attempt is made to compare their findings.

Number of individuals affected

In his 1965 study, Sheiham[9] found that the prevalence of chronic periodontitis was 50·5% for 15- to 19-year-olds,

75·9% for 20- to 24-year-olds and 100% for 55- to 59-year-olds. By contrast, in the 1978 United Kingdom survey of dentate adults,[7] the frequency of chronic periodontitis was substantially lower, from 3% of 16- to 24-year-olds to 65% of over-55-year-olds. One might, therefore, be tempted to conclude that a remarkable improvement in periodontal conditions had taken place between 1965 and 1978. However, the conflict between these sets of statistics may be readily explained by reviewing the respective examination criteria. Both studies diagnosed chronic periodontitis when pockets of 3 mm or greater were present, a yardstick of disease which, although hallowed by time, has never been adequately tested. In the UK survey,[7] teeth were investigated for chronic periodontitis by probing only if they exhibited intense gingivitis or a marked change in gingival contour. Since chronic periodontitis may be present without evidence of overt gingival inflammation (fig. 1), it is likely that the 1978 UK survey statistics[7] underestimate the true frequency of chronic periodontitis. In Sheiham's study,[9] a diagnosis of chronic periodontitis was made when pockets of 3 mm or greater were present around any tooth, presumably regardless of its gingival condition.

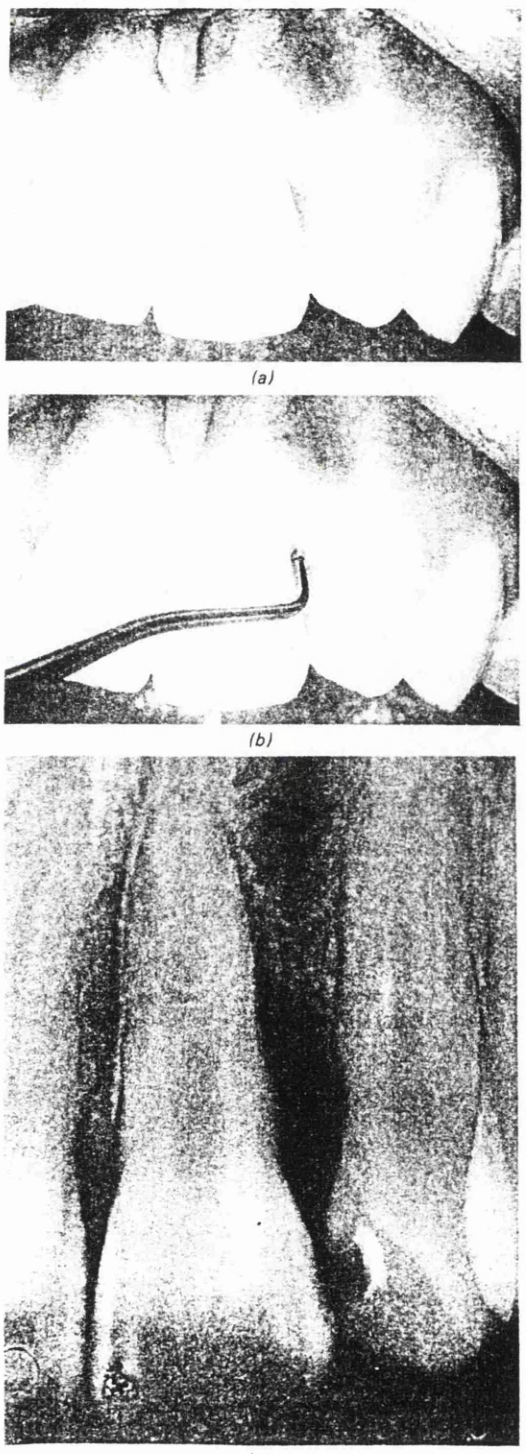

Fig. 1. Three views of the periodontal conditions around 12. (a) Apparently normal gingiva; (b) chronic periodontitis as detected with a probe; (c) radiographic evidence of marginal bone destruction.

Finally, prevalence figures for the existence of chronic periodontitis based solely on pocketing must be interpreted with caution, since it has been shown that the probe tip is likely to penetrate the pocket base to give an elevated score, the more easily if inflammation is present.[11] This caveat would of course apply to both studies quoted.

Number of teeth affected

In the 1968 England and Wales survey of dentate adults,[5] chronic periodontitis, identified by the existence of pockets of 3 mm depth or greater, affected only two teeth per person, on average. By contrast, using the same examination criteria, Sheiham and Dimmer[10] found that there were, on average, 10·2 pockets per person among a Northern Ireland factory population. The disparity in the results of these two surveys is difficult to explain although, in the Northern Ireland study, males outnumbered females by 5 : 1 and this may account to a small extent for the higher figure in the Irish study, since periodontal conditions are known to be worse in males than in females.[12]

The 1968 England and Wales survey[5] gave details of the distribution of gum conditions around the mouth, from which it may be calculated that for adults of all ages only 7·2% of first molar teeth exhibited chronic periodontitis, as identified by pockets of 3 mm or greater. This proportion of first molar teeth affected by periodontitis is slightly higher than that reported in the 1972 survey of dentate Scottish adults[6] where only 2% of first molars were judged to be affected by chronic periodontitis according to the same 3 mm criteria. Taken at face value, this statistic would suggest that periodontal disease is not a significant problem in Scotland, since it has recently been shown that pocket formation does not usually occur in other regions of the dentition without including one or more of the first molars.[3] It is not difficult, however, to understand why the 2% figure must be a gross underestimate if the examination criteria used in the Scottish survey are scrutinised. Only buccal and lingual marginal gingivae were examined. The condition of the interdental gingivae was ignored in spite of the fact that plaque, gingival inflammation and periodontal pockets in

adults are observed most frequently on approximal surfaces[13] and periodontal disease progresses faster interdentally.[14] Pockets were not probed unless the buccal or lingual marginal gingiva was 'markedly red or bluish red in contrast to the colour (usually pink) of adjacent healthy tissue'. Minor inflammatory changes were presumably to be ignored and inevitably a large number of periodontal pockets would be missed. The examiners, once they had identified marked gingivitis in buccal or lingual marginal gingiva, were instructed to confine their probing to that part of the tooth circumference and not to look for pockets in the papillary area.

This low frequency of chronic periodontitis, and therefore of marginal bone loss, around first molars in adults of all ages, should be viewed in the light of two reports of radiographic studies[15,16] which suggest that, even by the age of 14 years, the prevalence rates for marginal bone loss adjacent to first molar teeth are 51·5%[15] or 44%.[16] However, the validity of the criteria used to denote bone loss in these studies has been questioned.[17]

The severity of chronic periodontitis

Most of the major epidemiological studies throughout the world have reported the prevalence and severity of periodontal disease in terms of mean severity scores.[12] As a result, it is now well established that periodontal disease experience within any particular community depends to a large extent on prevailing standards of oral hygiene. Such preventive health practices are largely determined by social factors and, as a result, it is not surprising to find that, in numerous studies throughout the world, periodontal disease experience in groups of the same age, sex, socioeconomic and educational status is broadly similar.

Mean severity scores, owing to considerable data smoothing, do not describe the pattern of periodontal destruction, that is, the differences in severity of periodontitis between different teeth or between individuals. For example, the periodontal index (PI),[18] used widely in epidemiological studies, is based on a non-linear scale of 0, 1, 2, 6, 8 in which the criteria for scoring are weighted heavily in favour of destructive periodontal disease. For example, a mean score of 2 may be obtained from individuals with totally different clinical conditions such as 20 teeth with circumscribing gingivitis (score 2) or five out of 20 teeth with advanced destructive periodontal disease (score 8).

In United Kingdom populations only limited data are available concerning the degree of periodontal destruction. In the 1972 Scotland survey,[6] it was reported that only 0·7% of all teeth in all ages were so severely affected by periodontal disease that they should be extracted. The independent surveys in Wales,[8] England[9] and Northern Ireland[10] have presented data in mean severity scores in terms of the PI and are in general agreement, showing that the PI increases from about 1·3 in 15- to 19-year-olds to about 3·6 in 30- to 34-year-olds and 5·2 in 50- to 54-year-olds.

While there is fairly close agreement between PI surveys of UK populations, these studies show that periodontal disease experience in the United Kingdom may be very much greater than in American white populations where the PI increased from about 0·5 in 20-year-olds to about 2·0 in 70-year-olds.[19] In Sweden, too, periodontal conditions appear to be substantially better where the PI ranges from about 2·0 in 30- to 35-year-olds to about 3·5 in 61- to 65-year-olds.[20] Examiner variability in the use of the PI, however, may be responsible for some of the differences in periodontal status observed in these international comparisons.

Conclusions

Comparison of epidemiological data from United Kingdom surveys reveals major inconsistencies in the reported prevalence of chronic periodontitis. These inconsistencies can be traced to a lack of uniformly applied diagnostic criteria.

The conflict between epidemiological studies of chronic periodontitis is by no means confined to the United Kingdom. Comparison of data on the occurrence and distribution of periodontal disease throughout the world reveals the same inconsistencies, which Page and Schroeder[21] related to a lack of agreement on the clinical and radiographic recognition of periodontitis.

Data on the severity of periodontal disease in the United Kingdom are virtually limited to mean severity scores which do not describe the pattern of periodontal destruction. Nevertheless, comparison of these data with studies in America and Sweden in which the same index of disease was used, reveals that United Kingdom populations may have substantially more periodontal disease.

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Radiographic Assessment of Periodontitis

A Study of 800 Unreferred Patients

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Radiography may be used to assess the cumulative effect on marginal bone of a lifetime experience of periodontitis. An orthopantomographic survey of marginal bone loss in a Glasgow population of 800 dental out-patients is described: 84·5% of 16- to 19-year-olds exhibited marginal bone loss, and 4·6 teeth per person on average were affected. In all the older age groups the proportion of affected individuals was between 95·9% and 100% and, in the 45-and-over age-group, 81·1% of teeth present were affected. Severity data are also reported, revealing substantial interindividual variations in the pattern of destruction. Although many individuals, especially in the older age-groups, had teeth affected by advanced degrees of destruction, it seems likely that the great majority of teeth were probably amenable to periodontal treatment of some sort.

Chronic periodontitis is an inflammatory lesion affecting all the periodontal structures. It results from the apical extension of an inflammatory gingival lesion. Destruction of the periodontal attachment is accompanied by marginal bone loss and the formation of a periodontal pocket.

In a recent literature review[1] of chronic periodontitis in United Kingdom populations, major inconsistencies were noted in the reported prevalence of chronic periodontitis. These inconsistencies were traced to a lack of uniformly applied clinical diagnostic criteria. This paper discusses the rationale of radiographic methods of periodontal diagnosis and reports the findings of a radiographic study of 800 adults from a population of dental outpatients in Glasgow.

Radiographs can impart information on periodontal disease which is either difficult or impossible to obtain by the clinical approach. They can demonstrate the occurrence of periodontal disease by its destructive effect on supporting bone and reveal not only the amount of bone destroyed but also the amount remaining. Radiographs cannot reveal whether the bone loss is associated with current disease or with tissues which have been restored to health by treatment.

Only interdental bone may be visualised; facial and lingual bone is almost completely obscured by the radiodensity of the superimposed tooth. On the other hand, it is well established that the main clinical manifestations of periodontal disease are observed most frequently on approximal tooth surfaces[2,3] and that attachment loss proceeds faster interproximally than buccally or lingually.[3] Perhaps a more serious weakness, inherent in the radiographic assessment of bone loss, is the tendency to over-estimate the extent of bony support when an interproximal crater is present, overshadowed by facial and lingual bone margins.

Epidemiological assessment of marginal bone loss has been based on apparent qualitative changes as well as on reduction of alveolar bone height.

The traditional radiographic signs of early periodontitis, prior to reduction in crestal bone height, are marginal widening of the periodontal membrane space and loss of surface continuity of the alveolar crest. These criteria were used by Hull *et al.*[4] and Davies *et al.*[5] in their bitewing radiographic investigations of English schoolchildren. A more recent study has shown that the integrity of the crestal lamina dura may be unrelated to visible inflammation, bleeding on probing, pockets, or loss of attachment,[6] thereby casting doubt on the significance of apparent qualitative changes in the radiographic image of alveolar bone.

The quantitative measurement of marginal bone loss introduces a degree of objectivity lacking in clinical

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indices and qualitative radiographic evaluation. Assessment of the amount of alveolar bone loss has been undertaken by direct millimetric measurement[4,5] and by a proportional principle,[11] relating the actual alveolar bone level to the optimum bone height.

It has been shown both histologically[7,8] and radiographically[9,10] that the alveolar bone margin of normal, intact periodontal tissues lies 1 mm apical to the amelocemental junction. Radiographic measurements of reduction in alveolar bone height from this norm have been used to determine the prevalence and severity of marginal bone loss.[11]

For periodontal diagnosis in clinical practice, the radiographic technique of choice is the long-cone paralleling method, which produces a life-size image of the tooth with excellent definition. Unfortunately, this technique is time-consuming and costly and is therefore inappropriate to epidemiological work. Panoramic radiography, in which the entire dentition may be examined with one exposure, is a simple, comfortable and comparatively inexpensive alternative which will subject the study population to minimal radiation.

Methodological studies[12-15] have shown that, in quantitative assessment of marginal bone loss, the orthopantomograph (fig. 1) compares reasonably well with intra-oral radiography. However, whereas intra-oral radiography will permit bone level measurements on virtually every approximal surface, orthopantomography will have a success rate of only 81% on average[15] because of blurring, low contrast and overlapping (fig. 1). This deficiency of the panoramic technique, although limiting its usefulness in clinical work, is nevertheless acceptable in epidemiological research.

The purpose of this survey was to obtain radiographic data on the prevalence and severity of marginal bone loss among casual attenders at Glasgow Dental Hospital and School.

Fig. 1. Orthopantomograph revealing most proximal surface bone margins. Note the typical overlapping of upper canine and premolar teeth.

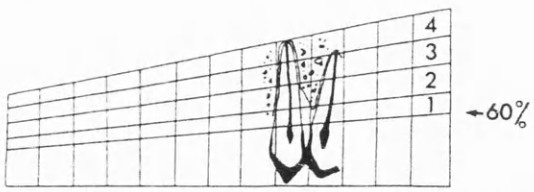

Fig. 2. Ruler for measuring bone loss. Calibration of ruler into four grades of bone loss. Grades 2, 3 and 4 are based on an optimum bone height of 65% while Grade 1, denoting the early stage of bone loss, is based on an optimum height of 60%, so as to avoid including borderline cases among pathological ones. The tooth illustrated gives proximal surface scores of Grade 1 (bone loss = 0-25%) on one surface and Grade 2 (bone loss = 25-50%) on the other.

Materials and methods

The study population comprised 800 unreferred dentate individuals of 16 years and over who presented for examination at Glasgow Dental Hospital and School during the period February to May 1978 as 'casual' patients. Pregnant women were excluded.

Individuals were questioned as they assembled to await diagnosis. Those who had at least one fully erupted tooth remaining were told of the purpose of the survey and invited to participate. The refusal rate was 1·0%.

A rotational tomographic view (fig. 1) was taken for each patient using the Siemens Orthoceph 3. Kodak X-Omat RP film (15 × 30 cm) in a Siemens Palomex cassette with super-high-speed intensifying screens was used at operating factors of 15 mA and 65–75 kV to record the images. Each radiograph was then duplicated to permit repeat evaluations to be made in the assessment of examiner variability.

All radiographs were assessed and data collected by one of the authors. The technique of radiographic assessment was similar to the method of Bjorn and Holmberg[15] who designed a transparent ruler to calculate the height of the alveolar bone in proportion to the total length of the tooth. Their ruler was calibrated so that bone loss could be scored in terms of 'quarters' of optimum bone height, which according to the investigation of Engelberger *et al.*[16] was considered to be 65 ± 5% of the total tooth length. However, to allow for variations in crown:root ratio and to avoid over-estimating the frequency of disease, they considered bone loss to be absent unless the bone level was less than 60% of total length (fig. 2).

All identifiable alveolar bone margins on mesial and distal surfaces of all teeth were marked with a felt-tip pen together with their crown tips and root apices. In the case of the multirooted maxillary molars, the buccal root tips were marked. Each approximal surface was assessed by applying the ruler over the radiograph (fig. 2) with its coronal and apical baselines coincident with the crown tip and root apex respectively. The ruler was inclined so that the long axis of the tooth was parallel to a line perpendicular to the coronal baseline. The bone loss score for each proximal surface was obtained according to the position of the bone margin within the ruler grid system. Where a line was located exactly over the bone margin reference mark, the lower score was recorded.

Intra-examiner variability was assessed by repetition of the marking and measurement procedure on 80 radiographs (10% of the total) selected at random from the collection of duplicate radiographs.

Tooth scores and approximal surface scores were analysed. The tooth score was obtained by selecting the greater of the two approximal surface scores. Thus a tooth with mesial bone loss of Grade 1 and distal bone loss of Grade 2 (fig. 2) received a score of 2. The tooth scores, therefore, represented the greatest amount of bone loss identifiable on radiograph for each particular tooth.

Tooth surfaces for which the bone margin could not be

Table I. Age, sex and social class distribution with average number of teeth present

Age-group (years)	Number of participants						Social class distribution						No. teeth present
	M + F		M		F		I	II	III	IV	V	Other	
	No.	%	No.	%	No.	%	%	%	%	%	%	%	
16–19	155	19·4	69	14·2	86	27·3	2·6	7·9	49·7	23·9	12·3	4·5	25
20–24	195	24·4	115	23·7	80	25·4	4·1	11·3	42·6	20·0	18·5	3·6	25
25–29	117	14·6	74	15·3	43	13·7	4·3	13·7	42·8	24·8	12·8	1·7	24
30–34	90	11·3	52	10·7	38	12·1	6·7	7·8	40·0	30·0	13·3	2·2	23
35–39	67	8·4	49	10·1	18	5·7	3·0	4·5	46·3	23·9	16·4	6·0	21
40–44	68	8·5	52	10·7	16	5·1	5·9	5·9	41·2	20·6	25·0	1·5	20
45 +	108	13·5	74	15·3	34	10·8	1·9	13·9	42·6	25·0	14·8	1·9	16
All ages	800	100	485	100	315	100	3·9	9·8	43·9	23·6	15·8	3·1	23

identified were treated statistically as though no bone loss was present, and teeth for which a reading could not be obtained at either approximal surface were also regarded as free from bone loss.

Results

Table I shows the age, sex and social class distribution, together with the average number of teeth present in each age group: 60·6% of the sample were male and males outnumbered females in each age group except the 16- to 19-year group. The modal age group was 20–24 years for males and 16–19 years for females; 23·7% of males and 27·3% of females fell within these cohorts. Out of the 155 individuals in the 16- to 19-year age-group, only 19 were 16 years of age. Of the 108 individuals in the 45-and-over age-group, only 11 were 60 years or over.

With regard to the social stratification, 43·9% were in Class III and the distribution of individuals within different social classes was broadly similar for all age groups.

The average number of teeth present fell from 25 in 16- to 19-year-olds to 16 in the 45-and-over group.

Owing to overlapping and poor contrast in certain regions of the dentition, it was not possible to identify the bone margin at all approximal tooth surfaces. Only 78·6% of approximal surfaces were measurable. However, 89·8% of teeth had at least one approximal surface where the bone margin could be identified, and all these teeth were given a bone loss score.

There was a wide variation in the frequency with which the mesial and distal surfaces of different tooth types could be measured. The upper canines and premolars were poorest in this respect. For example, the bone margin on the mesial aspect of the upper first premolar could be identified for only 13·5% of these teeth. The distal bone margin was identified for 41·2% of upper first premolars and bone loss scores for the tooth as a whole were obtainable for 44·6% of these teeth. For all other teeth, upper and lower, bone loss scores could be obtained for at least 90% of each tooth type.

Among the 80-patient subsample, 83·7% of tooth surfaces were scored the same on the two occasions they were measured, and 99·6% of surface scores did not differ by more than one grade when scored on two occasions.

Table II shows the number of individuals with evidence of marginal bone loss on at least one tooth surface. The prevalence of marginal bone loss was very high in both sexes: 84·5% were affected in the 16- to 19-year age-group and this rose to approach 100% by the late twenties. The prevalence rate for females was slightly lower than for males.

Table III shows the average number of teeth affected by marginal bone loss, which increased from 4·6 in the 16- to 19-year age-group to 14·1 in the 40 to 44-year age-group. This represents 18·3% of teeth present in 16- to 19-year-olds and 70·9% of teeth present in the 40- to 44-year age-group. In the 45-and-over age-group, 12·9 teeth on average were affected, representing 81·1% of teeth at risk.

Figure 3 shows three age-groups selected for the purpose of this paper to reflect the influence of increasing age on marginal bone loss. Thus, within each of the three

Table II. Prevalence of marginal bone loss by age and sex

Age-group (years)	Proportion with bone loss		
	Both sexes	Males	Females
	%	%	%
16–19	84·5	89·8	80·2
20–24	95·9	98·3	92·5
25–29	99·1	100	97·7
30–34	98·9	100	97·4
35–39	98·5	100	94·4
40–44	100	100	100
45 +	100	100	100
All ages	95·6	98·1	91·7

Table III. Distribution of teeth with various degrees of bone loss by age

Age-group years	Degree of bone loss									
	Under 25%		25–50%		50–75%		Over 75%		Any degree	
	Mean no.	No. affected as % of teeth at risk	Mean no.	No. affected as % of teeth at risk	Mean no.	No. affected as % of teeth at risk	Mean no.	No. affected as % of teeth at risk	Mean no.	No. affected as % of teeth at risk
16–19	4·4	17·4	0·2	0·8	0·02	0·07	0	0	4·6	18·3
20–24	6·4	25·8	0·5	1·9	0·01	0·04	0·005	0·02	6·9	27·5
25–29	8·1	33·9	1·4	5·8	0·1	0·6	0·03	0·1	9·7	40·4
30–34	8·5	37·1	2·6	11·2	0·3	1·2	0·07	0·3	11·4	50·7
35–39	9·2	43·9	3·3	15·6	0·4	1·8	0·06	0·3	12·9	60·6
40–44	8·6	42·9	4·7	23·2	0·6	3·2	0·3	1·3	14·1	70·9
45 +	6·5	40·9	5·0	31·0	1·1	7·2	0·2	1·2	12·9	81·1
All ages	7·0	30·7	2·0	8·8	0·3	1·3	0·07	0·3	9·3	41·0

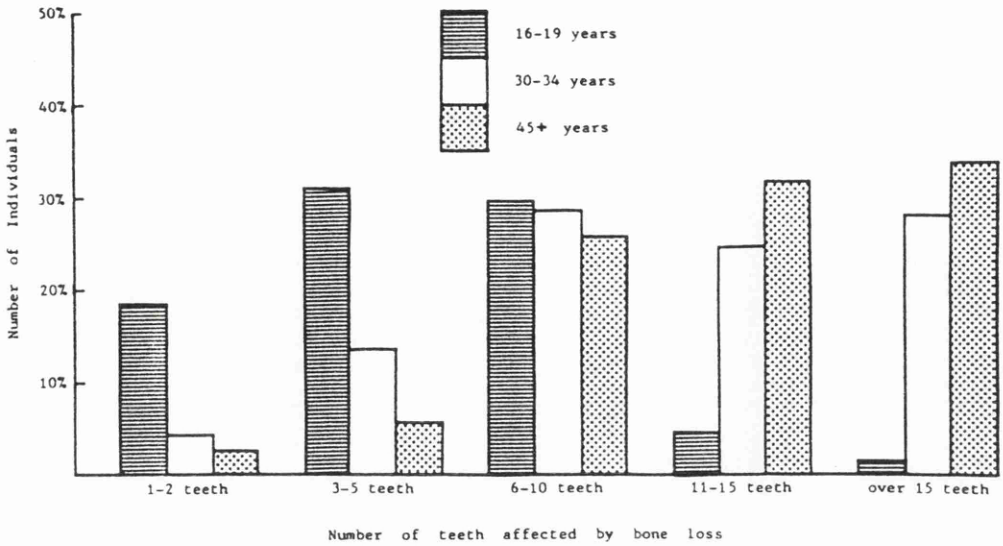

Fig. 3 Proportion of individuals with bone loss by the number of teeth affected

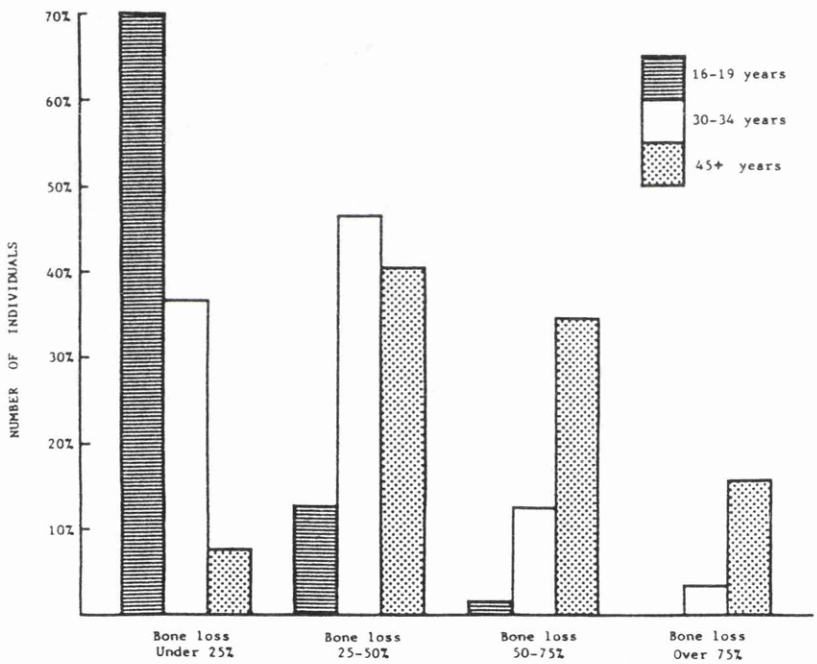

Fig. 4 Prevalence of different degrees of bone loss, that is, the number of individuals affected by worst affected tooth

selected age-groups, there was considerable variation in the number of teeth affected by bone loss, although to some extent this reflects variations in the number of teeth present.

The degree of bone destruction

Figure 4 shows the prevalence of different degrees of bone loss for the same three selected age-groups. In the 16- to 19-year age-group, only 1·3% of individuals exhibited marginal bone loss exceeding 50% of optimum bone height on at least one surface, and, in this cohort, bone loss exceeding 75% of optimum height was not found. However, the prevalence of the more advanced states of bone destruction increased with age so that 15·7% of the 45-and-over age-group had at least one tooth exhibiting more than 75% reduction in bone height on at least one surface.

Table III shows, for each age-group, the average number of teeth affected by different amounts of destruction. These figures reveal that, in 16- to 19-year-olds, bone loss exceeding 25% of optimum height was comparatively rare, occurring in only 0·87% of teeth at risk. On the other hand, in the 45-and-over age-group 39·4% of teeth present had suffered bone loss of this

degree. However, bone loss exceeding 50% was rather uncommon even in the older age-groups and was, in fact, noted in only 1·6% of all teeth in all ages.

Discussion

The results of this survey are based on the finding of marginal bone loss, which merely denotes a lifetime experience of chronic periodontitis rather than the presence of current disease. Marginal bone loss was assumed to be present when the alveolar bone height was less than 60% of total tooth length (fig. 2). If bone loss was present according to that criterion, it was graded on a severity scale of 1–4 representing bone loss in four quarters of optimum bone height (65% of total tooth length).

The validity of these criteria of bone loss is supported by the work of Hugoson and Koch[2] who showed radiographically that the average bone height among a hundred 15-year-olds (whom one would not expect to show widespread evidence of bone loss) was 63·6% of the total tooth length. Furthermore, it has been shown from approximal surface measurements of 320 extracted teeth (20 of each tooth type), assuming the normal bone margin to lie 1 mm apical to the amelocemental junction,[7-10] that 64·4% ± 3·78 (SD) of tooth length is normally invested with bone.[17] In the latter series of measurements, only 11·4% of approximal surfaces, because of relatively high crown:root ratios, appeared to have been originally covered with bone to less than 60% of tooth length. On the strength of these investigations it was felt that the technique of bone height measurement used in the present study would not over-estimate the prevalence or severity of marginal bone loss in a large population.

A bone loss score could be obtained for 78·6% of approximal surfaces. This corresponds closely with the measurability rate of 81% of tooth surfaces in Bjorn and Holmberg's methodological study[15] and indeed the distribution of measurable surfaces is similar in both studies. A major cause of non-measurability was overlapping of upper canine and premolar teeth due to unfavourable projection angles in those segments of the arch (fig. 1). Overlapping generally affected only the coronal half of the tooth so that, if substantial marginal bone loss existed, the bone margin could still be clearly seen in a zone where overlapping was absent. It is likely, therefore, that many of the teeth which could not be scored due to radiographic overlap were unaffected by major bone loss, since their bone margins coincided with the overlapped area in the region of the amelocemental junction.

Although it would be interesting to know which tooth types were most frequently affected, the distribution of bone loss around the mouth could not be judged with accuracy in this study, owing to the variations in measurability rate between different tooth surfaces.

There was a very high prevalence of marginal bone loss, even in the lower age-groups, and more teeth became affected with increasing age.

Bone loss exceeding 25% of optimum height occurred in only 0·87% (33 teeth) of teeth at risk in 16- to 19-year-olds (Table III). Nevertheless, these 33 teeth, as is evident from figure 4, were distributed among 14·7% (22 individuals) of the 16- to 19-year-old sample. As one might expect, the number of teeth with advanced degrees of bone destruction increased with age so that, in the 45-and-over age-group, 1·2% (20 teeth) of teeth present were affected by bone loss in excess of 75% (Table III). Figure 4 shows that these 20 teeth were distributed among 15·7% (17 individuals) of the 45-and-over sample.

The pattern which emerges, therefore, is of a disease which affected the majority of the population, but the number of teeth affected and the degree of destruction occurring in different individuals was subject to great variation.

Only 1·6% of all teeth in individuals of all ages exhibited a bone defect exceeding 50% of optimum height (Table III). Since one could argue that a tooth which retains at least 50% of its support should be amenable to treatment, very few teeth, by this crude yardstick, were affected beyond repair by periodontal disease.

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The 'high risk' group in periodontitis

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The high risk concept of periodontitis incorporates the theory that multiple tooth loss from periodontal disease affects a relatively small proportion of the population. This paper reviews and extends the evidence for such a concept by describing the occurrence and distribution of advanced bone loss in a Glasgow population of 800 dental out-patients. In common with other published studies of attachment loss or periodontal bone destruction, advanced bone loss was highly prevalent, affecting 54·1% of 50- to 73-year-olds. However, in parallel with other investigations, a small but with age increasing minority of patients accounted for most of the bone destruction. For example, in the 50- to 73-year-old age group, 28% of patients accounted for 75% of the advanced bone loss seen in that group.

It is well established that dental bacterial plaque is the primary aetiological agent of inflammatory periodontal diseases. It is equally well accepted that some individuals have a much greater risk of suffering severe and widespread periodontitis (inflammatory destruction of supporting tissues) than others. There is, however, less agreement as to the size of the 'high risk' group, a factor of considerable importance in health care planning. If the high risk group is very small, say 10% or less of the population, then some means of identifying susceptible individuals while still at an early stage of the disease may allow more efficient use of limited treatment resources.

Ideally, to calculate the proportion of teeth and individuals experiencing advanced destructive periodontitis, a longitudinal study is required in which the subjects are followed from puberty to old age. This is quite impracticable, of course, and conclusions must be drawn instead from cross-sectional studies or from the limited longitudinal work which is presently being undertaken.

The epidemiological data presently available to support the high risk concept of periodontitis come from several sources: measurements of attachment loss and pocket depth, radiographic measurements of marginal bone loss and studies on extracted teeth.

Löe *et al.*[1] carried out a 15-year longitudinal study of Sri Lankan tea labourers, initially aged 14–31 years, who were not accustomed to carrying out oral hygiene procedures. Periodontal destruction was widespread, due perhaps to racial factors or undernourishment as well as high plaque levels. Yet, it was still possible to identify a 'high risk' group of 8% in whom periodontal disease resulted in loss of virtually the entire dentition by 45 years of age.

Baelum *et al.*[2] reported cross-sectional findings in a population of adult Tanzanians, aged 30–69 years, who, like the Sri Lankan labourers (above) harboured abundant quantities of dental plaque. In contrast to the Sri Lankans, only 10% of tooth surfaces among the Tanzanians had advanced loss of attachment. Furthermore, relatively few individuals accounted for most of the advanced loss of attachment. Similar findings were reported in a more recent study of adult Kenyans.[3]

Another way of assessing the contribution of periodontal disease to tooth loss is to examine the periodontal condition of teeth in patients attending for full clearance. This has been done by dental panoramic tomograph assessment[4] and by direct measurement of loss of attachment on extracted teeth.[5] Thus, Linden[4] showed that among 373 individuals, only 15% had more than 50% of their teeth, with more than 50% reduction in bone height at the time of full clearance; 38% of patients 40 years and over had no teeth with this amount of bone loss. Similar findings in a Dutch community are reported by Bouma *et al.*[5]

All of the studies cited above point tantalisingly to the existence in most societies of a small group of individuals at high risk of losing a large number of teeth through periodontitis. Indeed, drawing on his own research findings and on other surveys in which pocket depths were used to describe disease severity, Schaub[6] concluded '. . . . that 10–15% of adult populations with natural teeth will lose teeth because of progressive periodontal disease and that approximately half of them will lose most of their teeth'.

In 1984, Jenkins and Mason[7] reported the findings of a dental panoramic tomograph study of marginal bone loss in 800 casual attenders at Glasgow Dental Hospital and School. It was observed that only 1·6% of all teeth in individuals of all ages exhibited bone destruction exceeding 50% of optimum bone height. In the present report, the results of that study are further analysed to describe the distribution of teeth in that category within the 800 patient population, as a contribution to the debate on the high risk concept of periodontitis.

Materials and methods

Details of the sample, research methods and examiner variability have been described previously[7] and only a summary is given here. The study population comprised 800 unreferred dentate individuals (485 males, 315 females) of 16 years and over. They presented with a variety of dental complaints to be examined at Glasgow Dental Hospital and School during the period from February to May 1978 as 'casual' patients. Table I allows comparison between the distribution of socio-economic class in the present study and in the random sample of dentate subjects who participated in a national survey of adult dental health in Scotland.[8] These

Table I Distribution of socio-economic classes in the present study and for comparison a random sample of dentate Scottish adults

	Socio-economic class					
	I	II	III	IV	V	Others
Present study ($n = 800$)	3·9%	9·8%	43·9%	23·6%	15·8%	3·1%
ADHS study ($n = 1232$)[a]	6·2%	16·6%	52·2%	16·2%	6·8%	2·2%

[a]Adult Dental Health Scotland study, data for dentate adults from Todd and Whitworth.[4]

Table II Distribution of patients by age showing (a) the number affected by advanced bone loss (ABL), (b) the number affected by generalised advanced bone loss (GABL) and (c) the total number of patients with the average number of teeth present. Figures in parentheses refer to the percentage of all patients in that age group studied

	(a) Patients with ABL	(b) Patients with GABL	(c)	
Age group	No. (%)	No. (%)	Total patients No.	Teeth present No.
16-19	1 (0·6)	0 (0)	155	25
20-24	3 (1·5)	0 (0)	195	25
25-29	5 (4·3)	0 (0)	117	24
30-34	14 (15·6)	0 (0)	90	23
35-39	18 (26·9)	0 (0)	67	21
40-49	34 (33·3)	4 (3·9)	102	19
50-73	40 (54·1)	4 (5·4)	74	15
All ages	115 (14·4)	8 (1·0)	800	23

two populations were broadly similar, except for an increased proportion of socio-economic class V subjects in the present study. Furthermore, the average number of teeth present in each age group was comparable to this random sample of Scottish adults.[8] A rotational tomographic view was taken for each patient, using the Siemens Orthoceph 3. The height of alveolar bone at each approximal surface was calculated with a transparent ruler, using the crown tip and root apex as reference points. The ruler was calibrated to score bone loss in 'quarters' of optimum bone height. A 'tooth' score was obtained by selecting the greater of the two 'approximal' surface scores. Tooth surfaces for which the bone margin could not be identified were treated statistically as though no bone loss was present.

The data presented in this paper relate to the prevalence within the 800-patient sample of bone loss exceeding 50% of optimum bone height, referred to subsequently as advanced bone loss (ABL).

Results

The age of the individual and the percentage of their teeth affected by ABL showed a highly statistically significant correlation (correlation coefficient = 0·36, $P < 0·0001$). There was a similarly highly significant negative correlation between the number of teeth present and the age of the patient (correlation coefficient = −0·55, $P < 0·00001$). Also, the percentage of teeth affected by ABL and the number of teeth remaining in the individual revealed a highly significant negative correlation (correlation coefficient = −0·6, $P < 0·00001$).

Table II shows the distribution by age group of patients within (a) the 800-patient sample, (b) within a subsample affected by advanced bone loss (ABL) and (c) within a further subsample affected by generalised advanced bone loss (GABL), defined as at least 50% of remaining teeth with advanced bone loss. Out of the 800-patient sample, 115 patients (86 males, 29 females) were identified with ABL, of whom eight patients were affected by GABL. In percentage terms, 14·4% were affected by ABL and 1% by GABL. The prevalence of ABL varied from 0·6% in the 16–19-year age group to 54·1% in the 50–73-year age category and GABL was absent until 40 years of age, when it occurred in 3·9% of the 40–49-year-olds and 5·4% of 50–73-year-olds.

Table III shows the proportion of individuals in each age group accounting for 75% of the observed advanced bone loss in that group. Thus, with increasing age an increasing proportion of individuals accounted for most of the advanced bone loss. However, even in the oldest age group, 50–73 years, this amounted to a relatively small proportion of individuals.

Due to overlapping and poor contrast in certain regions of the dentition, 21·4% of approximal surfaces in the 800-patient sample were unmeasurable. In the 115 patient subsample, the proportion of unmeasurable surfaces was only 15·7%. However, all but 10·2% of teeth in the 800-patient sample and all but 5·9% of teeth in the 115 patient subsample had at least one approximal surface where a score could be given.

Discussion

Various techniques have been employed in radiographic surveys to quantify marginal bone loss.[7,9,10] The present study used the proportional principle relating the actual bone level to the optimum bone height as described by Björn and Holmberg.[9] Other investigators have measured the amount of bone loss in millimetres, using the amelocemental junction as a fixed reference point.[10] The respective advantages and limitations are discussed elsewhere.[7,9,10] Although radiographs cannot be used in isolation to diagnose current disease, they can demonstrate the previous occurrence of periodontitis by its destructive effect on supporting bone.

Table III The proportion of patients within the 800-patient sample accounting for 75% of the total number of teeth with advanced bone loss

Age group (years)	Proportion
30-34	8%
35-39	15%
40-49	15%
50-73	28%

A bone loss score could not be obtained for 21·4% of surfaces in the 800-patient sample. This failure rate is comparable to those of other dental panoramic tomograph surveys of periodontal bone loss.[4,9,10] A major cause of non-measurability was overlapping of upper canine and premolar teeth, due to unfavourable projection angles in those segments of the arch. Overlapping generally affected only the coronal half of the tooth, so that, if substantial marginal bone loss existed, the bone margin could still be clearly seen in an area where overlapping was absent. It is likely, therefore, that many of the teeth which could not be scored due to radiographic overlap were unaffected by major bone loss, since their bone margins coincided with the overlapped area in the region of the amelocemental junction. Thus, the observed prevalence of advanced bone loss in this population is unlikely to be seriously affected by the failure to score all of the approximal surfaces. The reduced frequency of non-measurability in the 115-patient subsample with advanced bone loss supports this assumption.

The present study, in agreement with others,[11-13] shows a highly significant negative correlation between the percentage of teeth affected by advanced bone loss and the number of remaining teeth. This suggests that, despite underestimating the total number of teeth affected by advanced bone loss, due to absent teeth, it was still possible to identify the 'at risk' individuals by the condition of their remaining teeth.

Table II shows that advanced bone loss was highly prevalent in the older age groups in this population, in common with many other published studies of attachment loss or periodontal bone destruction. For example, Okamoto *et al.*[14] have recently reported prevalence data from a periodontal survey of a random sample of urban Japanese: the prevalence of attachment loss greater than 6 mm rose from 4% of 20–29-year-olds to 45% of 50–79-year-olds. Likewise, in a radiographic study of Swedish Dental School patients, Papapanou *et al.*[13] reported a high prevalence of advanced bone destruction (a bone level value greater than or equal to 6 mm from the amelocemental junction), increasing with increasing age to 83% of 75-year-olds.

These data invite comparison with prevalence data of deep pockets, as reported in studies utilising the Community Periodontal Index of Treatment Needs (CPITN).[15] Thus, the prevalence of deep pockets in 35–44-year-olds in 12 European countries varied from 2% to 18% of individuals. This is lower than the prevalence rates for advanced bone and attachment loss described above, presumably because gingival recession often occurs as periodontitis progresses, so that pocket depths tend not to reflect the full extent of periodontal destruction.[2]

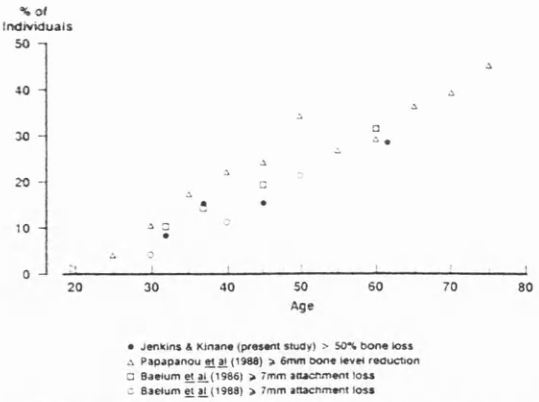

Fig. 1 Combination of data from four different studies on percentage of individuals in each age group accounting for 75% of the total number of sites affected by advanced periodontal destruction. Mid-points of age ranges are used and the criteria for determination of advanced periodontal destruction are as shown in the key to this figure.

While the prevalence of advanced bone loss was high in the present study, generalised advanced bone loss, that is affecting more than 50% of the remaining dentition, was uncommon. This affected only 3·9% of 40–49-year-olds and 5·4% of 50–73-year-olds. It must be remembered, however, that edentulous individuals were excluded from the present study, so that the low percentages recorded above may not accurately reflect the proportion of individuals in the general population who experience generalised advanced bone loss.

Another way of assessing how advanced bone loss is distributed within a population is to calculate the cumulative distribution of individuals according to the cumulative proportion of teeth with advanced bone loss. The data thus obtained in the present study (Table III) are in close agreement with the findings of other studies which have employed this method of analysis.[2,3,13] Figure 1 compares the results of the present survey with similar studies by Baelum *et al.*[2,3] and Papapanou *et al.*,[13] showing the proportion of individuals accounting for 75% of the total number of surfaces or teeth affected by advanced destruction in different age groups. Baelum *et al.*[2,3] carried out periodontal surveys of 170 adult Tanzanians and 1131 adult Kenyans, respectively, and their findings with respect to surfaces affected by at least 7 mm loss of attachment are recorded in figure 1. Papapanou *et al.*[13] carried out a radiographic survey of bone levels in a population of 531 Swedish Dental School patients and the data presented in figure 1 relate to proximal tooth surfaces with bone level values 6 mm or more from the amelocemental junction. Thus, figure 1 demonstrates a remarkable degree of agreement between all four studies, showing that a small, but with age increasing, minority of individuals account for most of the advanced periodontal destruction. At about 60 years of age, approximately 30% of individuals accounted for 75% of the advanced periodontal destruction in that age category.

In conclusion, the present study confirms the existence of a 'high risk' group in this population of unreferred Dental Hospital attenders and, by cautious extrapolation, in the Strathclyde Region of Scotland. However, the size of the 'high risk' group was somewhat larger than might have been

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anticipated from reports of epidemiological studies based on pocket depths. The present study, in common with others from various parts of the world, in which periodontal destruction was assessed by loss of attachment measurements or by radiographic assessment of marginal bone loss, demonstrated a high prevalence of advanced destruction, but also showed that relatively few individuals in each age group account for most of the advanced periodontal disease.

The finding that 28% of 50–73-year-olds accounted for 75% of the advanced destruction seen in that age group suggests that three-quarters of advanced periodontal disease could be prevented by targeting an effective preventive strategy on the 28% of individuals specially at risk. The question remains, whether an acceptable preventive strategy can be devised and whether it is possible to establish a simple method of identifying the 'at risk' group. At the present time there would appear to be no reasonable alternative but to advocate periodic thorough examination for all patients, treatment of all lesions while still at an early stage, and appropriate dental health education.

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Cross-sectional assessment of caries and periodontitis risk within the same subject

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Kinane DF, Jenkins WMM, Adonogianaki E, Murray GD: Cross-sectional assessment of caries and periodontitis risk within the same subject. Community Dent Oral Epidemiol 1991; 19: 78–81.

Abstract – Dental caries and chronic periodontitis may be synergistically associated, negatively associated, or completely independent. The present report examines this relationship by comparing the susceptibility to chronic periodontitis and caries within the same individual. From an 800-patient sample, a periodontitis risk score was derived by radiographic assessment of bone loss in quarters of optimum bone height and obtaining for each subject a mean score based on all measurable surfaces. Similarly the caries risk was determined radiographically from the total decayed and filled teeth (DFT), as a percentage of the total teeth measured. The Mantel-Haenszel technique was used for analysis of the relationship between periodontitis and caries and data was stratified on four categories of age, sex, and numbers of teeth present. This analysis revealed no systematic patterns, indicating that the risks of caries and periodontal diseases are unrelated ($\chi^2 = 0.00$; 1 df; $P > 0.50$). In addition, a regression analysis, which was controlled for sex and age, indicated a marked lack of association between caries and periodontitis ($P = 0.94$). Thus, although these common diseases share putative etiologic factors such as oral hygiene practices and dental attendance pattern, the major risk factors are probably quite different.

Key words: dental caries; periodontal disease; risk

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If the occurrence of dental caries and of chronic periodontitis are related it may be feasible to predict periodontitis by examining caries experience. Although the relationship between dental caries and chronic periodontitis has been addressed by many previous workers, their conclusions have been contradictory. In a study of Down's syndrome patients, an inverse association between periodontitis and smooth surface caries was found (1). In another selected patient group with juvenile periodontitis fewer proximal caries lesions were detected than in the control group without periodontitis (2). While these two studies suggest a negative relationship between caries and periodontitis for these specific disease groups, their findings would not be applicable to the general population. A negative association between caries and periodontitis was also reported by SEWON *et al.* (3) who compared the caries level of periodontitis-free individuals with a periodontitis-affected group in a cross-sectional study. This study, unfortunately, is flawed in that the authors selected and contrasted, on an *a priori* basis, two groups which they designated "periodontitis-free" ($n = 291$) and "advanced periodontitis" ($n = 125$), from an original population of 1105 Finns. They excluded individuals with mild bone loss or with less than six teeth and those for whom no sex and age matched controls existed. It is not clear from the disparate numbers in the designated groups how they arrived at matched pairs, and this study tends to pose more questions than it answers.

In contrast to the above findings, SKIER & MANDEL (4) examined the periodontal status of caries resistant and caries susceptible individuals in 40 sex and age matched pairs and found no significant differences between the two groups. However, they did find that female caries resistant subjects had a lower level of periodontitis and stated that this difference may have arisen because of the small numbers examined. This study is then somewhat equivocal and, although adequately designed, suffers from a lack of subjects. Although AINAMO *et al.* (5) looked at a large population (3344 Finnish military conscripts) and found no relationship between caries and periodontal disease within the same individual, the mean age of subjects was only 20.3 yr. Thus a high periodontal disease prevalence would not be expected.

In summary, the published research on this subject suffers from major shortcomings: none of the studies cited above has involved a sufficient number of subjects spanning a large age range which is representative of the general population. JENKINS & KINANE (6) have recently reported the results of a radiographic study of 800 unreferred dental outpatients (aged 16–73 yr) which determined a high risk group for periodontitis in several age categories of patients. The present report extends that study by comparing the susceptibility to periodontitis and caries within the same individual.

Materials and methods

Study population

Details of the sample, research method, and examiner variability have been described previously (7) and only a summary is given here. The study population comprised 800 unreferred dentate indi-

viduals (485 males, 315 females) 16 yr and over. They presented with a variety of dental complaints to be examined at Glasgow Dental Hospital and School during the period from February to May 1978 as "casual" patients. Comparison between the distribution of socioeconomic class in the present study and in the random sample of dentate subjects who participated in a national survey of adult dental health in Scotland (8) has been reported previously (6). These two populations were broadly similar except for an increased proportion of socioeconomic class V subjects in the present study. Furthermore, the average number of teeth present in each age group was comparable to this random sample of Scottish adults (8). This sample may be cautiously regarded as representative of the dentate Scottish adult population.

Methods

A rotational tomographic view was taken for each patient using the Siemens Orthoceph 3. The height of alveolar bone at each approximal surface was calculated with a transparent ruler using the crown tip and root apex as reference points. The ruler was calibrated to score bone loss in "quarters" of optimum bone height. Tooth surfaces for which the bone margin could not be identified were treated statistically as though no bone loss was present. A periodontitis risk score was derived by assessing bone loss in quarters of optimum bone height and obtaining for each subject a mean score based on all surfaces to which a bone loss score had been attributed. Similarly the caries risk was determined radiographically from the total of decayed or filled teeth (DFT) as a percentage of the total teeth measured.

$$\text{Periodontitis score} = \frac{\text{cumulated quarters of bone loss}}{\text{total measurable surfaces}}$$

$$\text{Caries score} = \frac{\text{filled and carious teeth}}{\text{total measured teeth}}$$

Due to overlapping and poor contrast in certain regions of the dentition, 21.4% of approximal bone surfaces in the 800-patient sample were unmeasurable for periodontitis. Similarly, in assessing caries experience 8.2% of the teeth were unmeasurable.

Statistical analysis

The Mantel-Haenszel technique (9) was employed in the statistical analysis of the data to allow for the possible confounders sex, age and teeth present. This test is based on the formulation of multiple 2×2 tables relating caries to periodontitis, with the study population grouped into 16 strata, defined by age (four groups), sex, and "number of teeth present". Teeth present, caries, and periodontitis groupings were made using the median value for the entire population and dividing the groups into "above the sample median" and "below the sample median". The principles underlying this statistical method have been described in detail by BRESLOW & DAY (10). The Mantel-Haenszel test was run on an ICL

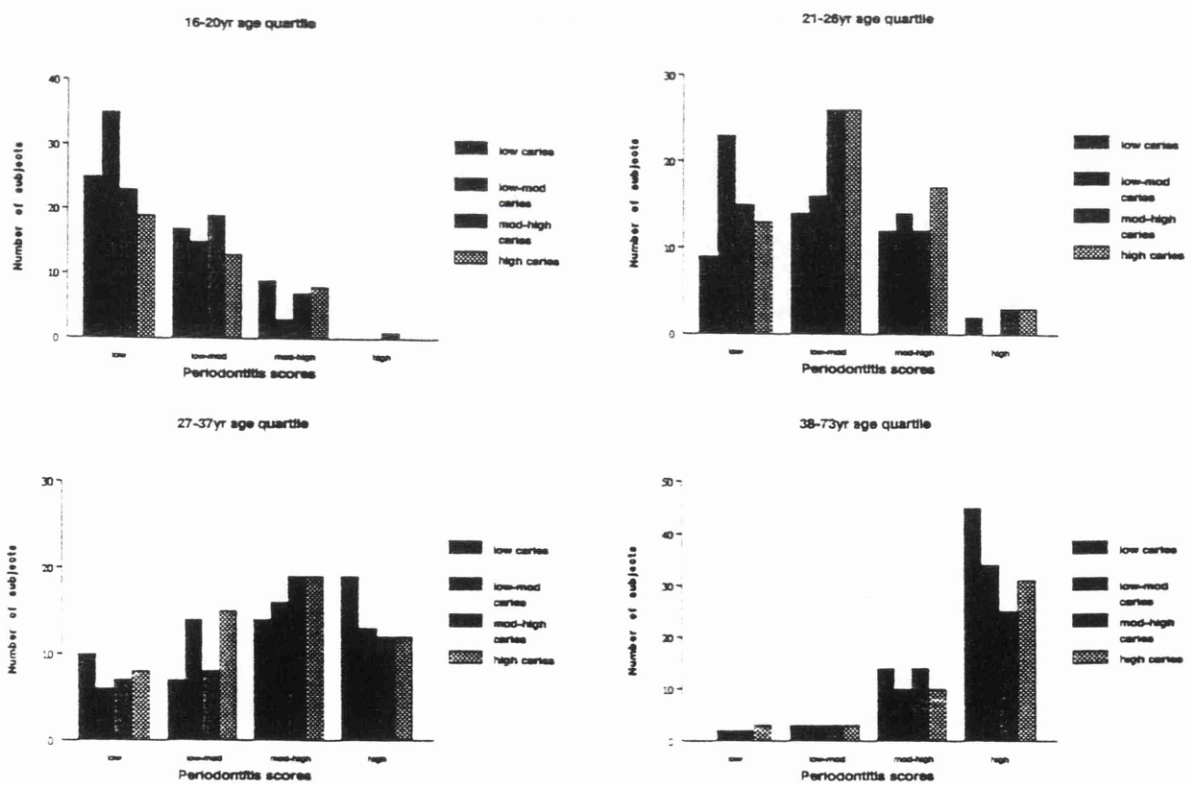

Fig. 1. A series of cross-tabulations of the grouped periodontitis and caries scores, performed separately for the four age quartiles.

VME mainframe computer using the BMDP statistical package.

Results

Analysis of the age and sex distribution of the study population indicates that males were on average 3 yr older than females and that the age distributions were slightly skewed towards the younger age groups. The distribution of caries scores by sex showed no differences between males and females.

In order to facilitate further analyses, caries and periodontitis scores were ranked in order of magnitude and divided into quartiles, which were then termed: mild; low to moderate (low-mod); moderate to high (mod-high); and high. A series of cross-tabulations of the grouped scores, performed separately for each age quartile (16–20 yr; 21–26 yr; 27–37 yr; and 38–73 yr) were constructed, but gave no suggestion of any association between caries and periodontitis when controlled for age (Fig. 1). Thus, Fig. 1 depicts the relationship between caries and periodontitis in the 16–20 yr age group and although periodontitis scores are relatively lower than in the other age groups, no pattern emerges for caries experience within the periodontitis severity groupings. A similar lack of pattern in caries and periodontitis experience is seen throughout Fig. 1, although the trend of increasing periodontitis experience with age is clearly evident.

The Mantel-Haenszel analysis relating periodontitis scores (above/below median) to the caries score (above/below median), stratified for sex (M/F), age (four categories), and teeth present (above/below median) indicated no relationship between caries and periodontitis ($\chi^2 = 0.00$; 1 df; $P > 0.50$). The calculated overall odds ratio for caries and periodontitis is 1.01 with 95% confidence intervals of 0.69 and 1.47. A test for comparison of cross-product ratios within the 16 strata indicated homogeneity within these stratifications ($\chi^2 = 7.19$; 15 df; $P = 0.95$).

The Mantel-Haenszel analysis allows for stratification and thus balances the effects of several potential confounders, i.e., age, sex, and number of teeth present, without requiring assumptions to be made about the data. However, it does have the disadvantage that it takes no account of the fact that both caries and periodontitis scores were measured on continuous scales and so a series of regression analyses were performed to examine the associations between the periodontitis score, caries score, age and sex. The periodontitis scores for males and females were markedly skewed and a log transformation was employed prior to subsequent analysis. Regression analysis revealed a highly significant association between age and periodontal disease ($R^2 = 47.3\%$, $P < 0.001$). A modest but statistically significant association between sex and periodontal score was also found ($R^2 = 2.9\%$, $P < 0.001$) but this association may have resulted from the difference in age distributions for female and male groups. A further analysis was performed to check this and revealed that age and sex were independently related to the periodontitis score. A regression analysis controlling for sex and age was then performed to determine any association between periodontitis and caries. This analysis returned a P value of 0.94, indicating a very clear cut lack of association between periodontitis and caries. A similar series of analyses to those carried out for the periodontitis index, were performed with caries as the response variable. These analyses showed no association between caries and "age", "sex", or "age and sex" and between caries and periodontitis controlling for "age and sex".

Discussion

A disadvantage of cross-sectional as compared to longitudinal studies is that missing teeth cannot be fully accounted for, i.e., the reason for extraction is unknown or unreliable. The Mantel-Haenszel technique allows for the possible effect of teeth missing without having to make assumptions about the data. This test enabled stratification of the data such that the possible confounders sex, age, and teeth present could be allowed for and also facilitated presentation. However, our data for periodontitis and caries experience were on continuous scales. Therefore, we also assessed the association between caries and periodontitis using a regression analysis, controlling for age and sex. This more sensitive analysis led us to an identical conclusion that there was no evidence of an association between caries and periodontitis experience.

Thus our results differ from others where a negative or positive association between caries and periodontitis within the same individual was suggested (1–4). Studies reporting a negative association between caries and periodontitis are largely based on selected patient groups with a recognised increased susceptibility to periodontitis (1, 2). The only study not using a periodontitis susceptible group which found a negative association was that of SEWON *et al.* (3) but this was based on a relatively small sample (115 pairs) from an uncharacterized population. The present study confirms the tentative conclusion of AINAMO *et al.* (5), which was made on a young population, and SKIER & MANDEL (4) that caries and periodontitis within the same individual are not related. If there is no association between the risks of caries and periodontitis, we cannot, therefore, predict that individuals with minimal caries should have more or less periodontal destruction than others. Thus dental primary health care workers should be vigilant in their assessment of both diseases and be wary of the lack of association in the susceptibility to these diseases. Thus, although these common diseases share putative etiologic factors such as oral hygiene practices and dental attendance pattern, the major risk factors are probably quite different. Longitudinal studies in which the reasons for each tooth lost are known would fully test this hypothesis.

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Longitudinal study of untreated periodontitis

(I). Clinical findings

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Jenkins WMM, MacFarlane TW and Gilmour WH: Longitudinal study of untreated periodontitis: (I). Clinical findings. J Clin Periodontol 1988: 15: 324–330.

Abstract. The principal aim of this study was to investigate the use of certain clinical and microbiological criteria to predict periodontal breakdown during a 1-year period. A further aim was to establish whether the act of collecting subgingival plaque samples periodically throughout the observation period would have any effect on the clinical or microbiological variables. Only the clinical data is presented in this paper. The study population comprised 11 volunteers (aged 32–51 years) with persistent advanced periodontitis and inadequate plaque control in spite of a previous intensive course of hygiene therapy. From the left jaw quadrants, 89 teeth were selected, yielding 148 bleeding pockets of 4 mm depth or more. From the right jaw quadrants, 74 teeth were selected, yielding 117 bleeding pockets of 4 mm depth or more. All subjects were examined on 7 occasions at 2-monthly intervals when plaque index scores, dichotomous measurements of gingival redness, pocket depths and attachment levels were recorded. Bacteriological sampling was carried out at each visit for each site only in the left jaw quadrants while the right jaw quadrants were sampled only at the first and last visits. During the study, no subgingival instrumentation was performed, except at 3 sites which exhibited loss of attachment of 3 mm. These teeth were withdrawn from the study for ethical reasons. At the completion of the study, the sequential changes in probing attachment level at each site were subjected to regression analysis to determine the direction and extent of attachment change which had taken place at each site. Further analysis revealed that neither the plaque index scores, the presence of gingival redness nor the pocket depth measurements could be used in a predictive capacity with respect to attachment loss. Comparison of right and left jaw quadrants showed that the act of sampling at 2-monthly intervals had no effect on any of the variables studied.

Key words: periodontitis: untreated; progression; clinical observations.

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For many years, a major goal of periodontal research has been the discovery of "prognostic indicators" which would allow the most susceptible patients and teeth to be singled out for priority treatment. Longitudinal studies during which periodontal status was monitored by traditional clinical means, however, have shown that pre-existing attachment loss, pocket depth, gingival condition, plaque accumulation, suppuration, or bleeding on probing could not be used to predict which sites would subsequently exhibit progressive attachment loss (Listgarten & Levin 1981, Haffajee et al. 1983a, Lindhe et al. 1983).

Alternatively, it has been suggested that various specific bacteria or bacterial morphotypes found in samples from periodontal pockets might indicate a susceptibility to destructive periodontitis (Listgarten & Levin 1981, Claffey et al. 1985, Slots et al. 1985).

The present study was carried out in an attempt to further elucidate the possible role of clinical and bacteriological criteria in predicting future attachment loss. A further aim was to establish whether the act of collecting subgingival plaque samples periodically throughout the observation period would have an effect on the clinical or microbiological variables. This article is concerned with the clinical findings. The microbiological data is presented in a separate paper in this issue (MacFarlane et al.).

Material and Methods

Subjects

11 patients with generalised advanced periodontitis volunteered to take part in this study. Approximately 3 months prior to recruitment, each patient had completed an intensive course of scaling, root planing and oral hygiene instruction with little evidence of clinical improvement: there was no reduction in pocket depth or in the number of bleeding sites and plaque control remained inadequate. It was, therefore, assumed that this group of patients who were 'resistant' to therapy might be subject to progressive periodontal breakdown and would form a suitable population in which to investigate the possible association between certain clinical and microbiological variables and subsequent attachment loss throughout a 1-year period when no subgingival instrumentation was carried out. The 11 subjects comprised 4 males and 7 fe-

males aged 32 to 51 years (mean age 40.7 years). All the participants were in good general health and had not received antibiotic therapy in the previous 6 months.

Selection of study sites

The selection of gingival sites was made at a recruiting appointment before the baseline visit. Bleeding pockets of 4 mm depth or more were selected which were sufficiently accessible for accurate clinical measurements to be made and for bacteriological sampling of subgingival plaque to be carried out. The precise location of each site was determined at the baseline visit and recorded on stone study models which had been obtained from impressions taken at the recruiting visit. A total of 89 teeth yielding 148 'test' sites were selected from the left jaw quadrants together with 74 teeth, yielding 117 'control' sites in the right jaw quadrants.

Experimental design

Fig. 1 summarises the experimental design. Following their agreement to participate, all subjects were examined on 7 occasions at 2-monthly intervals. With the exception of plaque and gingival redness, scores for which baseline measurements were not obtained until the second visit (due to a last-minute change in protocol), the clinical data was recorded at each visit for each selected site. Bacteriological sampling, however, was carried out at each visit for each selected site only in the left jaw quadrants. The right jaw quadrants were sampled only at the first and last visits. It was intended that left (test) and right (control) jaw quadrants should be compared to assess whether the act of sampling at 2-monthly intervals had any effect on the clinical or bacteriological variables. During the study, no subgingival instrumentation was performed but scaling and polishing was carried out to remove supragingival calculus and stain as necessary from any part of the dentition so affected. All the data collection and bacteriological sampling was carried out by the same individual.

Plaque index (PII)

All selected surfaces were scored according to the method of Silness and Loe (1964) on a scale of 0–3.

Gingival redness (GR)

Dichotomous measurements of gingival redness (presence or absence of colour change) were made at each selected site. Bleeding on probing at the pocket orifice was not assessed because of possible interference with subgingival plaque in shallow pockets.

Probing depth (PD)

Measurements were made at each selected site to the nearest millimetre using a Hu-Friedy PCP 12 probe with a tip diameter of 0.4 mm.

Attachment levels

Attachment levels were recorded to the nearest millimetre at each selected site by measuring the distance between the gingival margin and a fixed reference point on the visible tooth surface (e.g., a cusp tip, a restoration margin or the cemento-enamel junction) and adding this measurement to the corresponding pocket depth. The gingival margin to reference point measurements were made on stone models obtained from alginate impressions taken at each visit. This method was preferred to the construction of an acrylic stent because of obvious occlusal instability among several of the subjects. It was felt that tooth migration and over-eruption would render the stent ill-fitting by the end of the study.

visit	1	2	3	4	5	6	7
time (months)	0	2	4	6	8	10	12
plaque index		×	×	×	×	×	×
gingival redness		×	×	×	×	×	×
pocket depth	×	×	×	×	×	×	×
attachment level	×	×	×	×	×	×	×
bacteriology (left)	×	×	×	×	×	×	×
bacteriology (right)	×						×

Fig. 1. Experimental design.

Collection of samples

Supragingival plaque was removed using a curette and discarded. A clean curette was introduced into the pocket as far apically as possible and the bacterial contents removed. The samples were suspended in 0.2 ml sterile anaerobic blood broth and transferred as quickly as possible for laboratory analysis.

Determination of 'improving' or 'deteriorating' sites

Significant changes in attachment levels were identified in two ways.

(1) *During the study*
A loss of attachment of 3 mm or more was taken to be beyond the limit of measurement error and, therefore, reasonably good evidence of loss of periodontal support (Haffajee et al. 1983b). During the course of the study, all sites exhibiting a loss of attachment of 3 mm or more were given appropriate treatment in the form of scaling and root planing and were withdrawn from the study although the clinical and bacteriological data obtained up to that point were used in the statistical analysis.

(2) *Retrospectively*
At the completion of the study the sequential changes in probing attachment level at each site were subjected to regression analysis using a method similar to that of Goodson et al. (1982). The projected change in attachment level (ΔY) was computed for each site. The ΔY values were used to identify for each patient the 3 sites exhibiting the greatest projected attachment loss and the 3 sites exhibiting the greatest attachment gain. The mean ΔY was also computed for each patient.

Individual sites were also classified into 1 of 5 categories representing the direction and extent of attachment change which had taken place over the one-year study period. To be classified as an 'improving' site the ΔY had to exceed $+1$ mm/year. Two grades of 'improving' site were created: a grade I site should exhibit a slope significantly different from 0 at the 5% level; whereas the slope of grade II sites would not differ significantly from zero. Likewise, to be classified as a 'deteriorating' site, the ΔY had to exceed -1 mm/year. 2 grades of 'deteriorating' site were creat-

ed according to whether the slope of the regression line differed significantly (grade V) from 0 at the 5% level or did not (grade IV). Stable sites (grade III) were those where the ΔY lay between +1 and −1 mm/year and none of these sites had slopes which differed significantly from zero. Thus, a spectrum of attachment change was created ranging from grade I, where there was good evidence of attachment gain, to grade V where there was equally good evidence of attachment loss. This analysis was performed to observe the site distribution of attachment level changes and to determine whether any correlations existed between the clinical and microbiological variables on a site by site basis.

Statistical analysis and data handling

The data for all patients was entered on to a disk file on an ICL 2988 mainframe computer, and the MINITAB statistical package (Ryan et al. 1985) was used to analyse the data.

Rank correlation was used to assess whether there was any relationship between mean ΔY values and: (1) the baseline mean PD, PII and GR scores of each subject; (2) the mean PII and GR scores of each subject over visits 2–7. Furthermore, the mean values of these variables were compared for the 3 'best' and 3 'worst' sites of each subject using paired *t*-tests and Wilcoxon signed-ranks tests. This approach allows the subject rather than the site to be taken as the experimental unit (Imrey 1986).

In addition, the site by site variation was examined by comparing mean levels of PD, PII and GR for the 5 attachment change grades using a one-way analysis of variance F test, after logarithmic transformation of the data if necessary (Armitage 1971).

Results

Attachment level changes

During the 1-year observation period, 5 sites in 4 subjects lost 3 mm of attachment. 3 of these 5 sites were withdrawn during the study for ethical reasons at the 4-, 8- and 10-month observation periods, while the other 2 sites did not accumulate 3 mm loss of attachment until the final visit. A further 21 sites in 6 subjects lost 2 mm attachment. Therefore, a total of 26 sites in 6 subjects exhibited attachment loss of 2 mm or more between baseline and final measurement. This data is reported for descriptive purposes only and to enable comparison with other studies. To obtain a more reliable estimate of disease progress, the whole series of attachment level measurements was subjected to regression analysis. This was thought to be a more sensitive method of identifying attachment changes for subsequent data handling.

Regression analysis yielded a projected change in attachment level (ΔY) for each site studied during the year of observation. On the test side, the results varied from a projected attachment loss of 9 mm/year (for the site which was withdrawn at 4 months) to a projected gain of 2.4 mm/year. On the control side, the ΔY varied from a loss of 3.6 mm/year to a gain of 2.4 mm/year. The site distribution of attachment level changes is presented in Table 1. The subjects have been ranked according to each individual's overall experience of deterioration or improvement as measured by their overall mean ΔY values. Taking all 265 test and control sites together, 169 (64%) sites were judged to have been stable (grade III) during the year of observation. Improving sites numbered: grade I, 16 (6%); grade II, 15 (6%), while deteriorating sites numbered: grade IV, 33 (12%); grade V, 32 (12%). The frequency and distribution of attachment level changes was similar in both test and control sites.

In comparing the behaviour of different patients' dentitions, it is apparent that the deteriorating sites were fairly widely distributed. Only 3 subjects, A, B and C, had no sites in the deteriorating categories. 13 of the 31 'improving sites' were found in one subject, A, the only subject among the study group who received antibiotic therapy during the study. Penicillin was prescribed by the subject's doctor for a sore throat 1 week before the 8-month observation period and this was thought to explain the improvement in clinical variables which gathered momentum at 10-month and 1-year observation periods.

PD, PII and GR as predictors of deterioration

Subjects were ranked according to their mean ΔY value (most improvement = rank 1; most deterioration = rank 11) and also according to their baseline mean PD (shallowest = rank 1), PII and GR scores (lowest = rank 1). Rank correlation of the mean ΔY value of each subject with baseline mean PD, PII, and GR scores gave the following results for test and control sites respectively: mean

Table 1. No. of sites by grade of attachment level changes

Subject	mean ΔY (mm/year)	Grade I		Grade II		Grade III		Grade IV		Grade V		Total	
		test	control	test	control	test	control	test	control	test	control	test	control
A	−0.94	4	4	2	3	8	5	0	0	0	0	14	12
B	−0.35	1	0	0	0	7	8	0	0	0	0	8	8
C	−0.32	0	2	1	0	5	8	0	0	0	0	6	10
D	−0.02	1	1	2	1	11	8	2	2	1	1	17	13
E	−0.35	0	0	0	0	8	8	2	0	1	2	11	10
F	−0.43	1	0	0	0	9	8	1	1	3	2	14	11
G	−0.47	0	0	0	0	12	8	1	0	2	3	15	11
H	−0.57	0	0	1	1	6	4	1	1	2	3	10	9
J	−0.65	0	0	2	0	11	5	2	6	2	1	17	13
K	−0.70	1	0	1	1	7	9	8	2	3	2	20	14
L	−0.94	1	0	0	0	9	4	3	1	3	1	16	6
total		9	7	9	6	93	76	20	13	17	15	148	117
total all sites		16		15		169		33		32		265	

Note: subjects ranked A–L in order of increasing tendency towards deterioration.

PD (+0.01 and −0.21), mean PII (+0.05 and −0.04) and mean GR (+0.44 and −0.07). None of these correlation coefficients are significantly different from zero and there is no evidence of a consistent relationship between the mean amount of deterioration over 12 months and the baseline mean PD, PII and GR.

To investigate whether real associations were being masked by the inclusion of data from a large number of stable sites, the 'best' 3 and 'worst' 3 test and control sites were compared for each subject. Table 2 shows the baseline mean PD, PII and GR scores for the 'best' 3 and 'worst' 3 test and control sites of each subject. When these means were compared using paired *t* tests, there were no significant differences between the 'best' sites and the 'worst' sites nor were significant differences to be found between test and control sites. These results were unaltered when Wilcoxon's signed-ranks test was used instead of the paired *t*-test.

This lack of association was confirmed when the baseline mean levels of PD, PII and GR were compared for the 5 attachment change grades of test and control sites, treating the sites as independent units (data not shown).

Thus attachment level changes were not dependent on *initial* pocket depths, plaque or gingival redness. There was, furthermore, no appreciable difference between test and control sites with respect to initial pocket depths, plaque or gingival redness.

PII and GR over visits 2–7

Rank correlation of the mean ΔY value of each subject with mean PII and GR scores over visits 2–7 gave the following results for test and control sites respectively: mean PII (−0.08 and +0.38) and mean GR (+0.46 and −0.27). Although there is a suggestion of correlation here, between GR and loss of attachment, none of these correlations are statistically significant (e.g., rank correlation = +0.46, $n = 10$, $p = 0.17$).

Table 3 shows the mean PII and GR scores over visits 2–7 for the 'best' 3 and 'worst' 3 test and control sites of each subject. When these means were compared using paired *t*-tests, there were no significant differences between the 'best' sites and the 'worst' sites nor were significant differences observed between test and control sites. These results remained unaltered when Wilcoxon's signed-ranks test was used instead of the paired *t*-test.

This lack of association was confirmed when the mean PII and GR scores over visits 2–7 were compared for the 5 attachment change grades of test and control sites, treating the sites as independent units (data not shown). Thus attachment level changes were not dependent on the *prevailing* level of plaque or gingival redness throughout the observation period. Furthermore, the act of sampling carried out at test sites on each occasion appeared to have no effect on plaque or gingival redness scores.

PII and GR in each subject at each visit

Tables 4, 5 show the mean PII and GR scores respectively for all sites in each subject at each visit. The slopes of the least squares linear regression lines of mean PII and mean GR scores on time have been computed for each subject. The subjects have been ranked according to these slopes and rank correlation was used to compare these rankings with the subjects' rankings on clinical improvement/deterioration. The rank correlation coefficients were +0.36 for PII ($p = 0.25$) and +0.53 for GR ($p = 0.09$). If subject A is excluded, the rank correlation for GR falls to −0.37 ($p = 0.26$). This subject, it must be remembered, finished a course of penicillin 1

Table 2. Baseline means of probing depth (PD), plaque index (PII) and gingival redness (GR) for the best 3 sites and the worst 3 sites in each subject

	Test sites						Control sites					
	best 3 sites (mean)			worst 3 sites (mean)			best 3 sites (mean)			worst 3 sites (mean)		
Subject	PD (mm)	PII (0–3)	GR (0,1)	PD (mm)	PII (0–3)	GR (0,1)	PD (mm)	PII (0–3)	GR (0,1)	PD (mm)	PII (0–3)	GR (0,1)
A	5.0	0.8	1.0	5.7	0.7	1.0	6.7	0.7	1.0	4.3	1.7	1.0
B	5.0	2.3	0.7	5.7	2.0	1.0	6.0	1.7	0.7	5.8	2.3	1.0
C	6.0	1.0	0.3	5.0	1.3	0.3	5.0	1.0	0.8	4.4	1.4	1.0
D	5.7	1.7	0.3	5.7	2.0	1.0	5.0	2.0	1.0	6.3	1.3	1.0
E	5.6	0.4	0.4	5.3	1.0	0.3	5.8	0.5	0.5	5.2	0.5	0.3
F	7.7	1.3	1.0	4.8	2.0	1.0	5.5	1.8	1.0	5.7	1.7	1.0
G	4.3	1.7	1.0	4.7	0.3	1.0	5.3	1.7	1.0	4.7	1.7	1.0
H	4.3	*	*	6.7	*	*	5.3	*	*	4.3	*	*
J	7.0	1.0	0.8	7.7	2.3	1.0	6.0	1.8	0.5	4.8	1.8	1.0
K	4.7	0.7	1.0	4.7	1.0	1.0	5.7	2.0	1.0	4.0	1.7	1.0
L	4.3	1.7	1.0	4.3**	0.8**	0.5**	4.7	0.7	0.7	4.7	0.7	1.0
mean	5.41	1.25	0.75	5.46	1.34	0.82	5.54	1.37	0.81	4.92	1.46	0.93

Paired *t*-test results:

(I) Comparing mean of 'worst' 3 sites with mean of 'best' 3 sites.

(a) Test sites. PD: $t = 0.12$, $p = 0.90$; PII: $t = 0.37$, $p = 0.72$; GR: $t = 0.71$, $p = 0.50$.

(b) Control sites. PD: $t = -2.11$, $p = 0.06$; PII: $t = 0.61$, $p = 0.56$; GR: $t = 1.35$, $p = 0.10$.

(II) Comparing mean of test sites with mean of control sites

(a) Best 3 sites. PD: $t = -0.35$, $p = 0.73$; PII: $t = -0.57$, $p = 0.58$; GR: $t = -0.66$, $p = 0.53$.

(b) Worst 3 sites. PD: $t = 1.49$, $p = 0.17$; PII: $t = -0.53$, $p = 0.61$; GR: $t = -1.48$, $p = 0.17$.

* Missing data.

** Mean values given for worst 4 sites, 2 of which tied for 3rd place.

Table 3. Mean plaque index (PII) and gingival redness (GR) scores over visits 2–7 for the best 3 sites and the worst 3 sites in each subject

	Test sites				Control sites			
	best 3 sites (mean)		worst 3 sites (mean)		best 3 sites (mean)		worst 3 sites (mean)	
Subject	PII (0–3)	GR (0.1)	PII (0–3)	GR (0.1)	PII (0–3)	GR (0.1)	PII (0–3)	GR (0.1)
A	1.0	0.6	0.4	0.4	0.7	0.8	1.6	0.7
B	2.1	0.9	2.1	0.8	1.6	0.6	1.8	0.9
C	1.1	0.5	0.8	0.8	0.6	0.8	1.0	0.7
D	0.8	0.2	1.8	0.8	1.3	0.4	1.5	0.9
E	0.6	0.2	1.2	0.3	0.4	0.1	0.5	0.1
F	1.5	0.9	1.5	1.0	1.8	1.0	2.0	0.9
G	1.4	0.8	1.0	0.9	1.8	0.9	1.7	1.0
H	*	*	*	*	*	*	*	*
J	1.2	0.8	2.6	0.9	1.7	0.7	1.5	0.6
K	0.3	1.0	1.1	0.8	1.8	1.0	1.5	1.0
L	1.4	0.9	0.6	0.7	1.1	0.6	0.8	0.9
mean	1.15	0.67	1.28	0.74	1.26	0.70	1.39	0.78

Paired *t*-test results:
(I) Comparing mean of 'worst' 3 sites with mean of 'best' 3 sites.
(a) Test sites. PII: $t=0.58$, $p=0.57$; GR: $t=0.84$, $p=0.42$.
(b) Control sites. PII: $t=1.08$, $p=0.31$; GR: $t=1.06$, $p=0.32$.

(II) Comparing mean of test sites with mean of control sites:
(a) Best 3 sites. PII: $t=-0.61$, $p=0.56$; GR: $t=-0.37$, $p=0.72$.
(b) Worst 3 sites. PII: $t=-0.46$, $p=0.65$; GR: $t=-0.65$, $p=0.53$.

* Missing data.

Table 4. Mean plaque index values from 2–12 months for individual subjects

	Months						Rank by PII 'improvement'
Subject	2	4	6	8	10	12	
A	1.0	1.0	1.1	0.9	1.1	1.3	9
B	2.1	1.7	1.9	1.8	2.0	1.8	4.5
C	1.3	0.9	0.9	0.6	0.9	0.5	1
D	1.7	1.6	1.0	0.9	1.5	1.3	2
E	0.6	0.4	0.9	0.3	1.1	0.7	10
F	*	*	1.3	1.0	1.4	1.1	4.5
G	1.5	1.2	1.1	0.9	1.0	1.2	3
H	1.1	0.9	1.4	1.2	1.0	1.0	6
J	1.5	1.9	1.5	1.4	1.7	1.8	7
K	1.6	1.8	1.7	1.6	1.8	2.1	11
L	1.8	1.4	1.3	1.3	1.7	1.8	8

* Missing data.

Table 5. Mean gingival redness scores from 2–12 months for individual subjects

	Months						Rank by GR 'improvement'
Subject	2	4	6	8	10	12	
A	0.96	0.65	0.85	0.50	0.50	0.31	1
B	0.81	0.81	0.75	0.69	0.94	0.81	9
C	0.69	0.75	0.56	0.75	0.63	0.50	3
D	0.80	0.70	0.63	0.43	0.70	0.60	2
E	0.38	0.05	0.14	0.19	0.33	0.00	5.5
F	*	*	0.79	0.79	0.79	0.84	10
G	1.00	0.91	0.94	0.79	0.91	0.82	4
H	0.77	0.73	0.96	0.71	0.81	0.57	5.5
J	1.00	0.84	0.81	0.69	0.92	0.92	8
K	0.83	0.47	0.41	0.74	0.71	0.79	11
L	1.00	0.96	0.96	0.92	0.88	0.96	7

* Missing data.

week before the 8-month observation period at which point his mean GR scores fell markedly and had not risen by the 10-month and 1-year observation periods.

Thus, with the exception of subject A, those subjects who suffered the greatest amounts of attachment loss during the 1-year of observation did not show a tendency towards greater mean PII or GR values with time nor did patients with a tendency towards clinical improvement show any trend towards lower mean PII or GR scores.

Discussion

The main purpose of the present study was to investigate whether periodontal breakdown might be related to pre-existing or prevailing clinical conditions or bacteriological findings. To test this hypothesis, it was important that the population studied should exhibit a sufficient incidence of clinically detectable ongoing disease.

By the 1-year observation period, 5 out of 265 sites had demonstrated loss of attachment of *more than 2 mm*, a rate of 1.9% of sites per year. By comparison, Lindhe et al. (1983) found that 1.9% of sites per year exhibited attachment loss of *more than 2 mm* in a 6-year study of 64 untreated Swedish subjects with mild to moderate periodontitis. The same authors reported that 3.2% of sites exhibited attachment loss of this same magnitude in a 1-year study of 36 untreated Americans with advanced periodontitis (Lindhe et al. 1983).

Taking as a less stringent criterion of periodontal breakdown, an observed loss of attachment of *2 mm or more*, 26 out of 265 sites in the present study fell into that category, a rate of 9.8% of sites per year. Corresponding annual rates of breakdown of *2 mm or more* in the above mentioned studies were 6.2% for Swedish subjects (Lindhe et al. 1983) and 9.1% for American subjects (Lindhe et al. 1983). In addition, Lang et al. (1986) found that only 3.1% of sites per year exhibited attachment loss of *2 mm or more* in a 2-year study of 1054 sites in 55 patients under recall maintenance following treatment of advanced periodontitis.

Thus, the incidence of periodontal breakdown reported in the present study is similar to, and in some cases greater than the incidence in other study populations.

Linear regression analysis was the

method chosen in the present study to identify 'improving' and 'deteriorating' sites (Goodson et al. 1982, Badersten et al. 1985) so that the relationship, if any, between disease 'activity' and other clinical and bacteriological variables could be investigated. This method allows the rate of change of attachment (the slope of the regression line) to be evaluated in conjunction with the variability of the longitudinal measurements as determined by the probability level of the slope. In the present study, grade I and grade V sites had a greater validity as 'improving' and 'deteriorating' sites respectively than grade II and grade IV sites since the former sites not only exhibited a projected attachment change of more than 1 mm per year but their slopes also differed significantly from zero at the 5 per cent level.

Much has been written recently concerning the use of site or subject as the experimental unit in periodontal research (Blomqvist 1985, Laster 1985, Imrey 1986). The general consensus favours the use of the subject as experimental unit since sites within subjects cannot be regarded as independent. In this study both approaches to analysis were used and the results obtained are very similar. This will not, of course, be universally true.

It might have been expected that the act of sampling subgingival plaque every 2 months during the one year of observation would have had a beneficial effect on the periodontium of the test sites. This proved not to have been the case at all, there being no major difference in the frequency of attachment level changes or in mean plaque or gingival redness scores between test sites which were sampled at each visit and control sites which were sampled at the first and last visit only. By contrast, Mousques et al. (1980), investigating the effect of sampling on periodontal conditions in 18 subjects, observed a trend towards lower plaque and gingival index scores and slightly shallower pocket depths, 42 days after the pockets were sampled. The authors, however, were inclined to attribute this to the subjects' improved awareness of oral hygiene rather than an effect of sampling itself.

The finding that attachment level changes were not predicted by *initial* plaque, gingival redness, or pocket depth scores is entirely in accordance with other reports (Haffajee et al. 1983a, Listgarten & Levin 1981, Lindhe et al. 1983). Furthermore, in this study, attachment loss occurred independently of the *prevailing* oral hygiene and gingival condition throughout the observation period. Although there is a suggestion of correlation between the prevailing level of gingival redness and deterioration in attachment levels when results are averaged over all sites for each subject, the correlations observed are not statistically significant. Furthermore, there is no association between prevailing levels of gingival redness and attachment change when the 'best' three sites are compared with the 'worst' 3 sites for each patient. Also, none of the significance levels quoted have been corrected to allow for the fact that multiple significance tests have been performed. Such a correction would considerably increase the p-values quoted and lessen the statistical significance.

While slight variations in plaque and gingivitis were recorded in each subject between visits, there was no consistent upward or downward trend either in subjects who showed an overall improvement in attachment levels or in those who suffered attachment loss.

In conclusion, this study of untreated periodontitis supports the findings of other workers that plaque, gingival inflammation and pocket depth measurements cannot be used in a predictive capacity with respect to attachment loss. Furthermore, the act of bacteriological sampling at 2-month intervals for one year had no effect on the progress of periodontal disease.

Zusammenfassung

Eine Langzeitstudie unbehandelter Parodontitis. (I). Klinische Daten

Mit dieser Studie soll vor allen die Anwendbarkeit gewisser klinischer und mikroskopischer Kriterien zur Voraussage parodontaler Lysis während eines Jahres geprüft werden. Weiterhin beabsichtigt man festzustellen, ob während der Versuchsperiode in regelmässigen Zeitabständen vorgenommene, subgingivale Plaqueabstriche klinische oder mikrobiologische Variablen beeinflussen können. In dieser Veröffentlichung werden lediglich die klinischen Daten dieser Studie behandelt. Das Probandengut bestand aus 11 Freiwilligen (im Alter von 32–51 Jahren), bei denen trotz der Teilnahme an einem Intensivkursus für Hygienetherapie, eine fortgeschrittene und sich immer weiterentwickelnde Parodontitis vorlag. 89 Zähne mit 148 blutenden Taschen mit einer Tiefe von 4 mm oder mehr, wurden in den linken Kieferquadranten ausgewählt und in den rechten, 74 Zähne mit 117 blutenden, 4 mm oder tieferen Taschen. Alle Probanden wurden in 2-monatlichen Abständen 7 mal untersucht, wobei die Beurteilungseinheiten (scores) des Plaque Index, die Ergebnisse dichotomer Messungen der Zahnfleischrötung, der Sondierung der Taschentiefen und der Attachmentniveaus registriert wurden. In den linken Quadranten wurden bei jedem Besuch an jeder "Seite" (auch "Stelle" genannt = Zahnoberflächen mit dazugehörendem Parodont) bakteriologische Stichproben entnommen, während in den rechten Quadranten solche Stichproben nur bei den ersten und den letzten Besuchen entnommen wurden. Während der Studie wurde keinerlei instrumentelle Behandlung vorgenommen, mit Ausnahme von 3 "Seiten", bei denen ein Attachmentverlust von 3 mm entdeckt wurde. Die an diesen Zähnen registrierten Ergebnisse wurden aufgrund der, aus ethischen Gründen vorgenommenen Behandlung, aus dem Ergebnismaterial der Studie gestrichen. Nach Abschluss der Studie wurden die Veränderungen des sondierten Attachmentniveaus einer jeden "Seite" durch eine Regressionsanalyse beurteilt, um Richtung und Grössenordnung des eingetretenen Attachmentverlustes einer jeden "Seite" feststellen zu können. Weitere Analysen zeigten, dass weder der Plaqueindex oder die Zahnfleischrötung, noch die Messungen der Taschentiefen imstande waren, den tatsächlich eingetretenen Attachmentverlust vorauszusagen. Der Vergleich zwischen den rechten und linken Kieferquadranten zeigte, dass die in 2-monatlichen Abständen stattgefundene Entnahme von bakterienen Stichproben keinen Einfluss auf die hier studierten Variablen hatte.

Résumé

Étude longitudinale sur la parodontite non traitée. (I) Données cliniques

Le but principal du présent travail était d'examiner l'utilisation de certains criteres cliniques et microbiologiques pour prédire une destruction parodontale sur une periode d'un an. Cette etude se proposait en outre de

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déterminer si le fait de prélever périodiquement des échantillons de plaque sous-gingivale pendant toute la période d'observation aurait une influence sur les variables cliniques ou microbiologiques. On ne trouvera dans cet article que les données cliniques. La population étudiée comprenait 11 volontaires (âges de 32–51 ans) qui présentaient une parodontite avancée persistante et chez qui la maîtrise de la plaque était insuffisante, bien qu'ils aient auparavant reçu un traitement hygiénique intense. Dans les quadrants gauches, 89 dents présentant 148 poches d'au moins 4 mm et avec saignement ont été sélectionnées. Dans les quadrants droits, 74 dents présentant 117 poches d'au moins 4 mm et avec saignement ont été sélectionnées. Tous les sujets ont subi à 7 reprises, à des intervalles de 2 mois, un examen au cours duquel les scores de l'Indice de Plaque, la présence ou l'absence de rougeur de la gencive, la profondeur des poches et le niveau de l'attache ont été enregistrés. Des prélèvements bactériologiques ont été pratiqués à chacune des visites sur chacun des sites du côté gauche, alors que, sur le côté droit, on ne faisait de prélèvement qu'à la 1ère et à la dernière visite. Pendant le cours de l'étude aucun traitement instrumental n'a été pratiqué, à l'exception de 3 sites où il se produisait une perte d'attache de 3 mm. Ces dents ont été retirées de l'étude pour raison éthique. A la fin de l'étude, les modifications successives du niveau de l'attache au sondage pour chacune des localisations ont été soumises à une analyse de régression pour déterminer la direction et la grandeur du changement de l'attache ayant pris place dans chaque localisation. Une analyse ultérieure a mis en évidence que ni les scores de l'indice de plaque, ni la présence de rougeur gingivale, ni la profondeur des poches ne pouvaient servir d'indicateur pour prévoir la perte d'attache. Une comparaison des quadrants gauches et droits a montré que le fait de faire des prélèvements tous les 2 mois n'avait pas d'influence sur les variables étudiées.

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Longitudinal study of untreated periodontitis

(II). Microbiological findings

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Abstract. 11 volunteer subjects with advanced chronic periodontitis participated in a 1-year longitudinal clinical and microbiological study. Subgingival plaque was collected at each of 7 visits from 148 pre-selected sites in the left jaw quadrants (test sites) and on the first and last visits, only from 117 sites in the right jaw quadrants (control sites). All sites were examined clinically at each of the 7 visits, and the microbiological markers investigated were the % spirochaetes and % black pigmented *Bacteroides* species in subgingival plaque. At the completion of the study, the sequential changes in probing attachment level at each site were subjected to regression analysis to determine the direction and extent of attachment change. Possible correlations between attachment change and % spirochaetes or % black-pigmented bacteroides were investigated using both individual sites and individual subjects. No significant differences were observed in either of the microbial variables between test and control sites. Possible correlations between the microbiological markers and attachment changes were investigated at baseline, at the 12-month visit and using the microbial data accumulated over all 7 visits. Significant differences were observed only at the 12-month visit when the % spirochaetes of both test and control sites were significantly lower in subjects showing the greatest improvement in attachment level. Overall, these results indicate that quantification of either spirochaetes or black-pigmented *Bacteroides* species cannot be used reliably to identify or predict disease-active sites.

Key words: periodontitis; untreated; progression; microbiological.

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It is now well-established that traditional clinical criteria are inadequate for determining active disease sites in periodontitis or for measuring the degree of susceptibility to future disease (Listgarten & Levin 1981, Haffajee et al. 1983, Lindhe et al. 1983). Since bacteria are involved in the aetiology of most, if not all forms of periodontal disease a number of workers have investigated the use of microbiological markers to assist in the diagnosis and clinical management of chronic periodontitis.

Dark-field microscopical techniques have been used most commonly and the use of these tests in periodontitis has been reviewed by Greenstein & Polson (1985). Generally, spirochaetes have been found more frequently and in higher numbers at diseased sites compared to healthy sites (Listgarten & Heilden 1978, Lindhe et al. 1980, Armitage et al. 1982). In addition, attempts have been made to correlate spirochaete counts with a number of clinical variables: the strongest relationship being between spirochaete counts and pocket depth (Rosenberg et al. 1981, Evian et al. 1982). In a longitudinal study, Listgarten & Levin (1981) showed that spirochaete counts could be used to predict which subjects would experience active periodontitis in the following 12 months.

Culture studies have also been used to assist in clinical diagnosis, and, a close relationship between percentages of *B. gingivalis, B. intermedius* and *A. actinomycetemcomitans* and disease-active periodontitis has been reported (Slots et al. 1985, 1986). However, no such correlations were found by Moore et al. (1983).

Due to the variability in rate of attachment loss in chronic periodontitis, longitudinal studies are more appropriate than cross-sectional investigations for relating microorganisms to disease activity. In addition, during the period of study, ideally, patients should receive no therapy. However, most previous microbiological investigations have been cross-sectional and, in the few longitudinal studies which have been performed, usually treatment of some kind has formed part of the experimental protocol. Therefore, the present longitudinal study was carried out to relate changes in probing attachment level to the % spirochaetes and black pigmented *Bacteroides* species in samples collected over a 1-year period from untreated patients with chronic periodontitis. A further aim was to establish whether the act of collecting subgingival plaque samples periodically throughout the observation period would have an effect on the microbiological findings.

Material and Methods

Subjects and experimental design

The subjects investigated and the experimental design of the longitudinal study were as described in the previous paper by Jenkins et al. (1988). 11 subjects with generalised advanced periodontitis volunteered to take part in the 1-year study. All the participants were in good general health and had not received antibiotic therapy during the past 6 months. Bacteriological samples were collected at each of the seven visits from pre-selected sites in the left jaw quadrants (test sites) and on the first and last visits only from the sites in the right jaw quadrants (control sites). The determination of "improving" or "deteriorating" sites, both during the study and retrospectively, was performed as described by Jenkins et al. (1988).

Collection of samples

All the bacteriological sampling was carried out by the same individual. Supragingival plaque was removed using a curette and discarded. A clean curette was introduced into the pocket as far apically as possible and the bacterial contents removed. The samples were suspended in 0.2 ml of sterile anaerobic blood broth (Gibco – Paisley, Scotland) by vigorously agitating the tip of the instrument in the fluid. Plaque samples were transferred to the laboratory as quickly as possible and disaggregated by aspirating and expelling the fluid 10–20 times through a disposable tuberculin syringe with a 25 gauge needle.

The method used for counting the % of spirochaetes in subgingival plaque was that of MacFarlane et al. (1986). This technique involved the combined use of negatively stained smears, dark field microscopy and an image analysing system. Smears were prepared by adding 2 μl of the plaque suspension and 4 μl of filter sterilised 10% Nigrosin (BDH Chemicals Ltd., Poole, England) towards one end of a previously cleaned glass slide. The solutions were mixed and then evenly smeared over about two thirds of the glass slide using the edge of a 22 mm glass cover slip. The smears were dried in air for 15 minutes then mounted in one drop of Harleco Synthetic Resin (Kodak Chemicals, Liverpool, England) with a 22 mm square coverslip.

Microscopy and counting

The smears were examined on a Leitz Ortholux microscope with a 12 v 100 w power pack using a darkground condensor (D 0.80 0.95), and a 630 × dry fluorite objective containing a D 0.80 funnel stop. A Newvicon television scanner was connected to the phototube of the microscope and to an image analysing Optomax III System (Analytical Measuring Systems, Saffron Walden, Essex, England). The field under study was displayed on a video monitor, the microorganisms appearing white on a black background. After setting the detector control, the total bacterial count for each field was obtained in seconds using the 'total count' facility on the Optomax III System. It was possible to check by eye what the image analyser was counting since a white 'flag' appeared on the television monitor screen next to each object being enumerated. Therefore, the operator uses his own visual acuity as the standard to which the machine is adjusted, and is able to monitor the level of non-bacterial particulate matter in each field. Fields which contained obvious particulate debris were not counted.

Since the analyser could not differentiate between spirochaetes and other bacteria it was necessary to count the number of spirochaetes by eye and to record these for each field. Duplicate smears were prepared from each sample and 30 fields per smear were counted. The Optomax III System was interfaced with an Apple II Europlus Computer allowing the results from each field to be stored on a floppy disc and printed on an Epson printer MX80F/T III (Sinshu Seiki Co. Ltd., Nagano, Japan).

The remainder of the sample was diluted 1:100 and 1:1000 in 1 ml volumes of sterile anaerobic blood broth (Gibco – Paisley, Scotland). Using a spiral plating machine (Don Whitley Scientific, Shipley, England) 50 μl of each sample were inoculated onto a brain heart infusion agar plate (Gibco – Paisley, Scotland) supplemented with 7.5% blood and 1 ml vitamin K haemin solution (Gibco – Paisley, Scotland). Plates were incubated for 7 days at 37 C in an atmosphere of 85% N, 10% H and 5% Co within an anaerobic chamber (Forma Scientific, Marietta, Ohio).

After incubation, using a Spiral System Counting Grid, (Don Whitley Scientific, Shipley, England) the total viable count and the number of black pigmented bacteroides colonies present in each sample, were counted. Wherever possible the whole plate was counted, but where this was not possible a segment of the plate was used. In order to balance any irregularities in sample deposition a similar segment on another part of the plate was also counted.

Statistical analysis and data handling

The data for all subjects was entered on to a disk file on an ICL 2988 mainframe computer, and the MINITAB statistical package (Ryan et al. 1985) was used to analyse the data. Rank correlation was used to assess whether there was any relationship between mean ΔY values (projected changes in attachment level) and: (1) the baseline mean % spirochates or % black pigmented bacteroides counts of each subject; (2) the mean % spirochaete or % black pigmented bacteroides counts of each subject at the 12-month visit; (3) the mean % spirochaete and % black pigmented bacteroides scores of the test sites of each subject over visits 1–7. Furthermore, the mean values of these variables were compared for the 3 'best' and 3 'worst' sites of each subject using paired t-tests and Wilcoxon signed-ranks tests. This approach allows the subject rather than the site to be taken as the experimental unit (Imrey 1986). In addition, the site by site variation was examined by comparing mean levels of % spirochaetes and % black pigmented bacteroides for the 5 attachment change grades defined below using a one-way analysis of variance F test, after logarithmic transformation of the data if necessary (Armitage 1971).

Results

Attachment level changes

A total of 148 test and 117 control sites were examined in the 11 subjects. Taking all 265 test and control sites together, 169 (64%) sites were stable (grade III during the year of observation), 31 (12%) improved (16 grade I and 15 grade II), while deteriorating sites numbered 65 (24%) (33 grade IV and 32 grade V). The frequency and distribution of attachment level changes were similar in both test and control sites. Before the end of the study 3 teeth (2 on the test side and 1 on the control side) exhibited loss of attachment of 3 mm and were withdrawn for treatment.

% spirochaetes and black pigmented bacteroides as predictors of deterioration

Subjects were ranked according to their mean ΔY value (most improvement = rank 1; most deterioration = rank 11) and also according to their baseline mean % spirochaetes and black pigmented bacteroides (lowest value = rank 1). Rank correlation of the mean ΔY value of each subject with baseline mean % spirochaetes and black pigmented bacteroides scores gave the following results for test and control sites respectively: mean % spirochaetes ($+0.22$ and $+0.39$); mean % black pigmented bacteroides (-0.11 and -0.03). None of these correlation coefficients are significantly different from zero and there is no evidence of a consistent relationship between the mean amount of deterioration over 12 months and the baseline mean microbiological values.

To investigate whether real associations were being masked by the inclusion of data from a large number of stable sites, the 'best' 3 and 'worst' 3 test and control sites were compared for each subject. Table 1 shows the baseline mean % spirochaetes and % black pigmented bacteroides scores for the 'best' 3 and 'worst' 3 test and control sites of each subject. When these means were compared using paired t-tests, there were no significant differences between the 'best' sites and the 'worst' sites nor were significant differences to be found between test and control sites. These results were unaltered when Wilcoxon's signed-ranks test was used instead of the paired t-test.

This lack of association was confirmed when the baseline mean levels of % spirochaetes and % black pigmented bacteroides were compared for the 5 attachment change grades of test and control sites, treating the sites as independent units (data not shown).

Thus, attachment level changes were not dependent on the *initial* % of spirochaetes or black pigmented bacteroides counts. There was, furthermore, no appreciable difference between test and control sites with respect to the microbiological data.

% spirochates and black pigmented bacteroides at the 12-month visit

Rank correlation of the mean ΔY value of each subject with mean % spirochaetes and % black pigmented bacteroides scores at visit 7 gave the following results for test and control sites respectively: mean % spirochaetes ($+0.64$ and $+0.88$) and mean % black pigmented bacteroides ($+0.04$ and -0.28). There was a significant correlation at visit 7 between low mean spirochaete levels and mean ΔY valves for both the control ($p=0.006$) and test ($p=0.04$) sites. However, none of the correlations for the % black pigmented bacteroides counts were statistically significant.

Table 2 shows the mean microbiological scores at the 12 month visit for the 'best' 3 and 'worst' 3 test and control sites of each subject. When these means were compared using paired t-tests, there were no significant differences between the 'best' sites and the 'worst' sites nor were significant differences observed between test and control sites. These results remained unaltered when Wilcoxon's signed ranks test was used instead of the paired t-test.

This lack of association was confirmed when the mean % spirochaete and black pigmented bacteroides scores at the 12 month visit were compared for the 5 attachment change grades of test and control sites, treating the sites as independent units (data not shown). Therefore, although rank correlation of the data yielded a significant relationship between mean spirochaete counts and the subjects with greatest mean improvement in attachment levels this result was not evident when the 3 'worst' and 3 'best' sites were compared, or when the microbiological data was analysed on a site specific basis.

Spirochaetes and black pigmented bacteroides throughout the 1 year of observation

Rank correlation of the mean ΔY value of each subject with the mean % black pigmented bacteroides scores accumulated at test sites over all 7 visits produced correlation coefficients of $+0.24$ with mean % spirochaetes and -0.12 with mean % black pigmented bacteroides. When the mean % spirochaetes and mean % black pigmented bacteroides of the 'best' 3 and the 'worst' 3 test sites were compared for each subject (Table 3), no significant differences were found.

This lack of association was confirmed when the mean levels of % spirochaetes and bacteroides obtained from the test sites over all 7 visits were compared for the 5 attachment change grades, i.e., analysing each site as an independent unit. However, a slight but not quite significant trend towards higher mean spirochaete counts in deteriorating sites was found.

Means of % spirochaetes and black pigmented bacteroides for each subject

Table 4 shows the mean % spirochaetes

Table 1. Baseline values of % spirochaetes and % black pigmented bacteroides (BPB)

	Test sites				Control sites			
	best 3 sites		worst 3 sites		best 3 sites		worst 3 sites	
Patient	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB
A	2.3	0.0	1.4	0.0	12.5	0.0	8.4	0.0
B	1.4	4.0	1.8	17.6	3.2	5.4	4.4	11.2
C	9.2	0.0	1.1	4.5	1.4	0.7	3.7	17.2
D	7.5	29.2	12.9	54.3	15.4	52.2	9.3	43.2
E	4.6	0.9	4.6	0.1	8.0	0.0	2.1	0.1
F	20.4	*	23.3	*	12.6	*	11.8	*
G	1.8	0.0	5.0	12.2	6.2	7.9	5.6	7.6
H	6.0	0.1	6.0	0.1	5.5	1.7	6.7	2.0
J	11.9	6.8	6.1	4.9	5.8	0.6	7.8	1.5
K	9.9	0.1	11.4	0.5	14.5	0.4	9.4	4.6
L	1.2	0.1	3.0	1.5	7.2	4.2	4.5	0.2
mean	6.93	4.12	6.96	9.57	8.39	7.31	6.70	8.76

Paired t-test results:
(I) Comparing mean of 'worst' 3 sites with mean of 'best' 3 sites.
(a) Test sites. SPIRO: $t=0.03$, $p=0.98$; BPB: $t=1.96$, $p=0.08$.
(b) Control sites. SPIRO: $t=-1.74$, $p=0.11$; BPB: $t=0.69$, $p=0.51$.

(II) Comparing mean of 'test' sites with mean of 'control' sites.
(a) 'Best' 3 sites. SPIRO: $t=-0.77$, $p=0.46$; BPB: $t=-1.29$, $p=0.23$.
(b) 'Worst' 3 sites. SPIRO: $t=0.19$, $p=0.86$; BPB: $t=0.40$, $p=0.70$.

* Missing data.

Table 2. % spirochaetes and % black pigmented bacteroides (BPB) at the 12-month visit

	Test sites				Control sites			
	best 3 sites		worst 3 sites		best 3 sites		worst 3 sites	
Patient	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB	mean % Spiro	mean % BPB
A	0.0	2.5	3.9	16.5	0.9	6.2	0.6	3.7
B	0.0	9.5	1.2	1.8	2.3	6.2	3.8	18.8
C	15.2	0.3	5.5	0.0	7.5	0.1	5.0	0.0
D	0.0	24.7	1.9	28.5	3.3	39.0	4.6	44.5
E	2.2	0.0	0.8	11.0	0.0	0.2	2.4	1.0
F	27.1	1.3	63.1	3.7	43.4	0.4	40.4	7.8
G	1.8	3.2	3.5	1.1	6.0	8.3	6.9	5.0
H	9.9	3.9	5.0	14.9	5.0	10.2	9.7	7.1
J	13.8	4.1	19.7	5.5	13.7	2.3	10.8	5.3
K	1.7	7.9	17.3	7.1	5.2	4.5	16.8	3.6
L	14.5	0.2	11.1	0.8	12.3	0.2	10.3	1.3
mean	7.84	5.24	12.09	8.26	9.05	7.05	10.09	8.92

Paired *t*-test results:
(I) Comparing mean of 'worst' 3 sites with mean of 'best' 3 sites.
(a) Test sites. SPIRO: $t=1.14$, $p=0.28$; BPB: $t=1.54$, $p=0.15$.
(b) Control sites. SPIRO: $t=0.80$, $p=0.44$; BPB: $t=1.25$, $p=0.24$.

(II) Comparing mean of 'test' sites with mean of 'control' sites.
(a) 'Best' sites. SPIRO: $t=-0.65$, $p=0.53$; BPB: $t=-1.15$, $p=0.28$.
(b) 'Worst' sites. SPIRO: $t=0.85$, $p=0.42$; BPB: $t=-0.23$, $p=0.82$.

Table 3. % spirochaetes and % black pigmented bacteroides (BPB) for all test site data accumulated during the 12-month observation period

	Best 3 sites		Worst 3 sites	
Patient	mean % spiro	mean % BPB	mean % Spiro	mean % BPB
A	4.7	5.2	6.8	4.4
B	6.3	1.4	5.2	11.8
C	7.0	16.7	15.2	19.9
D	10.9	1.7	2.9	3.6
E	3.0	2.0	2.4	3.8
F	21.6	*	36.1	*
G	1.2	4.5	6.9	4.2
H	5.1	5.8	3.9	3.7
J	16.3	4.1	11.5	3.3
K	9.3	5.4	14.3	7.8
L	9.4	11.5	7.8	14.7
mean	8.62	5.83	10.27	7.72

Paired *t*-test results.
Comparing mean of 'worst' 3 sites with mean of 'best' 3 sites.
SPIRO: $t=0.87$, $p=0.41$; BPB: $t=1.70$, $p=0.12$.

* Missing data.

at test sites for each subject at each visit. There was no evidence of a relationship between baseline mean % spirochaetes and each subject's subsequent experience of attachment loss. Furthermore, although there was considerable inter-subject variation in % spirochaetes, there was some evidence of consistency in individual mean spirochaete counts over the 1-year period of observation. An exception was subject A who received penicillin therapy just prior to the 8-month visit which lead to a marked reduction in spirochaete counts for the rest of the study. The slope of the least squares linear regression line of mean % spirochaetes on time was computed for each subject. The subjects were ranked according to these slopes and rank correlation was used to compare these rankings with the subjects' rankings on clinical improvement/deterioration (mean ΔY values). The rank correlation coefficient was $+0.72$ ($p=0.02$). Table 5 shows the test site means of % black pigmented bacteroides over all 7 visits. Very great variation was observed both between subjects and between visits within subjects and no recognisable pattern emerged. The rank correlation between mean ΔY values and slope of linear regression line of mean % black pigmented bacteroides on time was $+0.02$.

Discussion

The statistical analyses of the results of this study was designed to establish: (i) the effect of sampling at 2-monthly intervals on the subgingival microbiota, (ii) if there was a simple correlation between % spirochaetes or % black pigmented *Bacteroides* species and the tendency towards deterioration or improvement of individual sites or subjects and (iii) if these bacteriological variables could be used to predict attachment loss.

In the present study, the data were analysed using both the site and subject as the experimental unit, the latter now being the favoured method (Blomqvist 1985, Laster 1985, Imrey 1986).

No significant differences were observed in % spirochaetes between test sites (which were sampled at 2-month intervals) and control sites (which were sampled at the first and last visits only) when the data were analysed using either the site or subject as the experimental unit. This is consistent with the previous findings of Mousques et al. (1980) who reported that sampling caused only a transient decrease in spirochaete percentages lasting only one week and Magnusson et al. (1985) who described no systematic alterations in % spirochaetes when sampling was carried out on 9 occasions over a 32-day period.

The possibility of a correlation between % spirochaetes and the occurrence of attachment changes was investigated. The mean % spirochaetes were calculated both on a site specific basis and using the subject as the unit of statistical analysis. Significant differences were observed only at the 12-month visit when the % spirochaetes of both test and control sites were significantly lower in subjects who exhibited greatest gain in attachment. Since spirochaetes are not found in large proportions in shallow periodontitis – affected pockets (Rosenberg et al. 1981, Evian et al. 1982), it seems likely that the difference in spirochaete percentages observed was a function of reducing pocket depth and not of attachment change per se. This explanation would also account for the

Table 4. Test site mean % spirochaete counts from 0–12 months for individual subjects

Subject	Months							Rank by % spirochaete 'improvement'
	0	2	4	6	8	10	12	
A	6	10	12	12	1	1	1	2
B	2	3	6	11	5	5	1	5
C	5	10	9	7	3	4	10	3
D	15	14	13	16	12	5	3	1
E	4	3	1	4	4	4	2	4
F	20	24	25	20	30	20	38	11
G	3	4	1	3	3	4	3	7
H	8	7	6	6	4	7	10	8
J	11	14	18	6	17	15	16	9
K	12	12	15	17	14	13	12	6
L	4	6	7	7	9	15	12	10

Subjects ranked A–L in order of increasing tendency towards deterioration according to each individual's mean projected change in attachment level assessed by regression analysis.

Table 5. Test site mean % black pigmented bacteroides counts from 0–12 months for individual subjects

Subject	Months							Rank by % BPB 'improvement'
	0	2	4	6	8	10	12	
A	0	4	14	8	0	1	5	4.5
B	8	9	1	4	16	18	4	8.5
C	2	5	3	0	3	2	0	3
D	52	10	1	15	40	6	37	2
E	1	1	3	3	1	1	5	6
F	*	6	27	12	26	42	3	10
G	3	4	3	4	15	7	4	8.5
H	1	1	3	13	2	12	10	11
J	4	0	2	1	4	4	5	7
K	2	13	15	5	2	4	6	1
L	1	25	7	34	25	3	8	4.5

Subjects ranked A–L in order of increasing tendency towards deterioration according to each individual's mean projected change in attachment level assessed by regression analysis.

* Missing data.

trend towards slightly increased spirochaete counts with increasing tendency towards deterioration in both the 12-month data and the data accumulated over 7 visits.

The lack of any significant difference between the *baseline* counts of deteriorating, stable and improving sites would indicate not only that the presence of these organisms in individual sites cannot be used in a predictive capacity but also that no association exists on a site specific basis between spirochaetes and disease activity.

The lack of a simple correlation between the spirochaete proportions of improving, stable and deteriorating sites is reasonably consistent with the findings of other investigations. Claffey et al. (1985) investigated subgingival spirochaete counts at the end of a 1-year study of 7 subjects during which, sites were classified as deteriorating or improving on the basis of linear regression analysis. When spirochaete percentages from the two categories of sites were compared, no statistically significant differences were found. Listgarten et al. (1984, 1986) found that spirochaete counts in individual samples from deteriorated sites and pooled samples from stable sites were not significantly different. Slots et al. (1985) found that, while there was a significant association between spirochaetes and progressive loss of attachment, several active sites did not reveal these organisms. The only published data which supports the predictive value of % spirochaete counts is by Listgarten & Levin (1981) and Listgarten et al. (1986) who demonstrated that disease-susceptible *subjects* had significantly higher baseline percentage spirochaete counts (using plaque pooled from 6 sites per subject) than disease-resistant subjects. If more subjects had been investigated in the present study, it is conceivable that a trend may have emerged towards higher mean spirochaete counts in disease-susceptible subjects. However, we used our resources principally to observe the variations between a large number of diseased sites in a small number of individuals. What is clear is the need to interpret spirochaete counts, however derived, with considerable caution.

The results of the present study as they relate to spirochaete populations apply also in a similar fashion to black pigmented *Bacteroides* species. That is, % counts of these organisms could not be used to identify or predict disease-active sites or subjects. Sampling, furthermore, had no effect on the proportion of black-pigmented bacteroides present.

In previous culture investigations of subgingival plaque, black pigmented bacteroides isolates have been identified to species level although data for only a few species (usually *B. gingivalis* and *B. intermedius*) have been presented. Therefore, there is no information in the literature with which to compare the total black pigmented bacteroides percentage counts which were obtained in the present investigation. It is generally accepted that no single culture medium will be equally effective in isolating all strains of a particular bacterial species, let alone all species of a particular genus. In addition, there are a number of factors which could result in variations in the isolation rate of the same bacterial species among different researchers using similar media, e.g., the nature and concentration of blood and other growth factors added and the manufacturer of the medium used. Therefore, the estimation of black pigmented bacteroides species using a single medium must inevitably be an approximation. The bacteroides isolates were not speciated in this study since the intention of this study was to examine as many samples as possible using cheap and simple laboratory tests. However, we have isolated and identified *B. gingivalis* from sub-gingival plaque samples in other studies using the same culture medium and techniques employed in this investigation.

The results of the present study revealed wide variation in the counts between individual sites with time, between different sites in the same individual and between sites in different individuals (data not shown). However,

none of the variation could be related to disease activity.

A close relationship between *B. gingivalis* and the presence of chronic periodontitis (Zamoon et al. 1985) or disease-active periodontitis (Slots et al. 1986) has been reported. Therefore, it is possible that if the black pigmented isolates from this study had been identified a correlation between *B. gingivalis* and active sites may have resulted. On the other hand, the fact that Moore et al. (1983) failed to find a close relationship between either *B. gingivalis* or other black pigmented *Bacteroides* species and chronic periodontitis could be interpreted as supporting the results of this study.

A number of factors may be responsible for the failure of this investigation to demonstrate a significant relationship between microbiological tests and the identification or prediction of disease-active sites. The laboratory techniques used for enumerating spirochaetes are different from the dark field microscopy methods used by previous workers. However, there is no reason to believe that if the conventional method had been used, a different result would have been obtained, since there is good evidence that the technique described by MacFarlane et al. (1986) gives reproducible results. Spiral platers have been used successfully by other workers to dilute and inoculate cultures in microbiological investigations of subgingival plaque (Loesche et al. 1982) and again there is no reason to believe that the use of the more commonly used tube dilution method would have produced data which would have demonstrated a stronger association between % pigmented bacteroides and attachment changes.

Listgarten et al. (1984) offered a number of hypotheses to explain why *sites* with increased pocket depth did not demonstrate significantly more spirochaetes than those derived from the 6 deepest probing sites in stable areas of the mouth: (i) an alteration in the host response without a change in the subgingival flora; (ii) a qualitative alteration in the plaque flora not detected by the laboratory techniques used; (iii) relatively brief episodes of disease activity, which may be accompanied by brief qualitative changes in plaque flora that cannot be detected by infrequent sampling. The same hypotheses may also explain the negative nature of our results. Furthermore, since the laboratory tests used are relatively crude with inherent variables which are difficult to control accurately, it is unlikely that significant differences between samples from active and stable sites will be recorded consistently unless the differences are large. Since it seems likely that the microbial changes which are associated with the onset of active periodontitis are complex, it is perhaps not surprising that significant differences in microbiological variables were not demonstrated by the relatively crude techniques used both in this and other studies.

Zusammenfassung

Eine Langzeitstudie unbehandelter Parodontitis (II). Mikrobiologische Ergebnisse

11 freiwillige Probanden mit fortgeschrittener chronischer Parodontitis nahmen an einer 1-jährigen klinischen und mikrobiologischen Langzeitstudie teil. Bei jeder der 7 Einbestellungen des Probandengutes wurden an 148 vorbestimmten "Stellen" (sites) der linken Kieferquadranten (Test-Seiten) subgingivale Plaqueabstriche entnommen, und bei der ersten und letzten Einbestellung ebenfalls an 117 "Stellen" der rechten Kieferquadranten (Kontroll-Seite). Alle "Stellen" wurden an allen Untersuchungszeitpunkten klinisch beurteilt. Die ausserdem untersuchten mikrobiologischen "Markierungsparameter" bestanden aus dem prozentualen Anteil der Spirochäten und dem prozentualen Vorkommen der schwarz pigmentierten *Bacteroides* – Arten in der subgingivalen Plaque. Gegen Ende der Studie wurden die laufenden Veränderungen des sondierten Attachmentniveaus an jeder "Stelle" durch eine Regressionsanalyse beurteilt, um Richtung und Grössenordnung der Attachmentveränderungen feststellen zu können. An den einzelnen "Stellen" und bei den einzelnen Probanden wurden Attachmentveränderungen und prozentuales Vorkommen von Spirochäten oder schwarz pigmentierten Bacteroides auf eventuelle Korrelationen hin untersucht. Signifikante Unterschiede zwischen Kontroll- und Test-"Stellen" wurden bei den mikrobiologischen Variablen nicht beobachtet. Die Möglichkeit des Vorkommens von Korrelationen zwischen den mikrobiologischen Markierungsparametern und den Attachmentveränderungen wurde bei der Ausgangsuntersuchung, bei der Untersuchung nach 12 Monaten und durch Auswertung der antimikrobiellen Daten, die bei allen 7 Einbestellungen registriert worden waren, untersucht. Signifikante Unterschiede wurden nur bei der Untersuchung nach 12 Monaten beobachtet. Der prozentuale Anteil der Spirochäten der Test- und Kontroll-"Stellen" war bei den Probanden mit den deutlichsten Verbesserungen des Attachmentniveaus signifikant geringer. Ganz allgemein deuten diese Resultate darauf hin, dass das mengenmässige Vorkommen der Spirochäten und der schwarz pigmentierten *Bacteroides*, zu einer verlässlichen Idifikation oder Voraussage der Vorkommens krankheitsaktiver "Stellen" nicht angewendet werden können.

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Résumé

Etude longitudinale sur la parodontite non traitée (II). Donnees microbiologiques

Onze sujets volontaires présentant une parodontite chronique avancée ont participé à une étude longitudinale d'un an clinique et microbiologique. Des prélèvements de plaque sous-gingivale ont été pratiqués à chacune des 7 visites dans 148 localisations préalablement sélectionnées du côté gauche (sites test) et à la 1ère et à la dernière visite seulement dans les 117 localisations sélectionnées du côté droit (sites témoins). Un examen clinique de tous les sites a été pratiqué à chacune des 7 visites; les indicateurs bactériologiques étudiés étaient le proportion de spirochètes et celle de *Bacteroides* à pigment noir dans la plaque sous-gingivale. A la fin de l'étude, les modifications successives du niveau de l'attache au sondage pour chacun des sites ont été soumises à une analyse de regression pour déterminer la direction et l'ampleur du changement de l'attache. Les corrélations existant éventuellement entre le changement de l'attache et les proportions de spirochètes ou de bacteroides à pigment noir ont été recherchées en utilisant individuellement et les sites et les sujets. Aucune différence significative n'a été observée entre les sites test et les sites témoins pour aucune des variables microbiologiques. Les corrélations existant éventuellement entre les indicateurs microbiologiques et les changements de l'attache ont été recherchées à l'origine, à la visite de 12 mois et en utilisant les données microbiologiques accumulées pendant les 7 visites. Des différences significatives n'ont été observées qu'à la visite de 12 mois, où les pourcentages de spirochètes, aussi bien dans les sites test que dans les sites témoins, étaient significativement moins élevés chez les sujets ayant présenté la plus grande amélioration du niveau de l'attache. Dans l'ensemble, ces résultats indiquent que les quantités de spirochètes ou celles de *Bacteroides* à pigment noir ne peuvent pas être utilisées de manière fiable pour identifier ou pour prédire l'activité de la maladie dans les sites.

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Chapter 5

Classification and epidemiology

A classification provides a useful framework to view the spectrum of disorders which may be included in the term 'periodontal disease'. No classification, however, has yet been devised which has achieved wide acceptance. The one illustrated in Table 5.1 includes a wide range of established and proposed clinical entities some of which are considered in more detail in the text below.

PLAQUE-ASSOCIATED DISEASES

'Plaque-associated diseases' are those inflammatory conditions of the periodontium with bacterial plaque as the primary aetiological agent.

Simple gingivitis. This is the common form of chronic gingivitis where no identifiable modifying factors are present.

Complex gingivitis. This occurs when specific local or systemic modifying factors exist.

Reactive epulides. These are localized polypoidal gingival lesions which originate from inflamed gingiva, often from the supracrestal fibre attachment, and, therefore, from beyond the base of the gingival or periodontal pocket. They are assumed to result from the stimulating effect of dental plaque on a localized area of particularly sensitive connective tissue. Epulides may be sessile or pedunculated and can be classified as: the pyogenic granuloma (*see* Fig. 3.1), which is characterized by extensive endothelial and fibroblastic proliferation associated with a dense chronic inflammatory infiltrate; the fibrous epulis, consisting of dense fibrous tissue, occurring either *de novo* or due to maturation of a pyogenic granuloma; and the giant cell epulis, resembling a pyogenic granuloma clinically and histologically with the addition of large numbers of multinucleated giant cells. The reactive epulides are treated usually by local excision, as described in Chapter 11.

Table 5.1. *Classification of periodontal diseases*

Plaque-associated diseases
Gingivitis
Chronic
Simple gingivitis
Complex gingivitis
e.g. mouthbreathing gingivitis
pregnancy gingivitis
puberty gingivitis
leukaemic gingivitis
drug-induced gingival hyperplasia
hereditary gingival fibromatosis
HIV-associated gingivitis
Reactive epulides
Acute
Necrotizing ulcerative gingivitis
Periodontitis
Chronic
Adult periodontitis
Early onset periodontitis
prepubertal periodontitis
localized
generalized
juvenile periodontitis
localized
generalized
rapidly progressive periodontitis
Periodontitis associated with systemic disease
Acute
Periodontal abscess
Non-plaque-associated diseases
Traumatic
Occlusal trauma
Gingival recession
Infective
Acute herpetic stomatitis
Immunological
Desquamative gingivitis

Periodontitis. This is thought to comprise a family of related but distinct diseases that differ in precise aetiology, natural history and response to therapy. Whereas different forms of gingivitis are

differentiated according to the influence of modifying factors, periodontitis is classified, less convincingly, by age of onset and severity, since its precise aetiology is insufficiently understood. Nevertheless, periodontal literature is replete with references to different types of periodontitis.

Adult periodontitis. This is the commonest form of periodontitis. It is considered to have its onset during or after adolescence, to progress relatively slowly compared to early-onset forms, and is unlikely to become clinically significant until the mid-thirties. However, the rate of attachment loss may increase at any time.

In cross-sectional surveys of the general population, adult periodontitis is responsible for the majority of cases identified with advanced destructive disease. Adult periodontitis has not been linked to impaired host defence mechanisms.

Early-onset periodontitis. This comprises those forms of the disease which are characterized by advanced destruction in childhood, adolescence or early adulthood. Early-onset types of periodontitis have been linked to specific subgingival microfloras, to impairment of immune and inflammatory mechanisms, and to familial distributions. These associations are strongest for localized juvenile periodontitis (*see* Chapter 15).

Prepubertal periodontitis. This is a term used to describe severe periodontitis in deciduous and permanent teeth before puberty. There is considerable evidence that deciduous teeth are just as vulnerable to periodontitis as their permanent successors, and that periodontitis in the permanent dentition often starts before puberty. However, generalized advanced periodontal destruction with premature tooth loss is very rare in prepubertal children, and is invariably associated with underlying systemic disease, such as neutropenia, hypophosphatasia etc. It is this advanced form, accompanied by severe gingivitis, which has been described as 'generalized prepubertal periodontitis'. So-called 'localized prepubertal periodontitis', affecting relatively isolated teeth in apparently healthy children, and unaccompanied by severe gingivitis, may, as noted above, be quite prevalent. It is suspected that the more advanced cases of localized prepubertal periodontitis may herald the later development of juvenile or rapidly progressive periodontitis (*see* below).

Juvenile periodontitis. This occurs in adolescence (*see* Chapter 15). It has its onset around puberty, although there is mounting evidence that it may manifest itself earlier in the permanent

dentition and be associated with attachment loss in the deciduous dentition. It may occur in two forms. Localized juvenile periodontitis, in which only incisors and first molars are involved, is the commoner form. In generalized juvenile periodontitis, many teeth are affected other than incisors and first molars. Some authorities, however, do not recognize generalized juvenile periodontitis as a distinct entity, preferring to group adolescent cases of severe generalized periodontitis with those having their onset in early adulthood, and which are classifiable as 'rapidly progressive periodontitis'. The prevalence rates of juvenile periodontitis among adolescent caucasians and ethnic negroid races are approximately 0.1% and 2%, respectively.

Rapidly progressive periodontitis. This is a term used to describe severe generalized periodontitis of the early-onset type affecting young adults between 20 and 35 years (or between 12 and 35 years, if generalized juvenile periodontitis cases are included). There is no reliable epidemiological data on the occurrence of rapidly progressive periodontitis. It is thought, however, to be the commonest form of early-onset periodontitis and a prevalence rate of 1–2% for caucasians has been suggested. Clinical diagnostic criteria to differentiate rapidly progressive from generalized juvenile or adult forms of periodontitis have not been agreed, and it remains unclear whether rapidly progressive periodontitis should be regarded as a discrete clinical entity, as a late-onset manifestation of generalized juvenile periodontitis, or as a severe early-onset variant of adult periodontitis.

Periodontitis associated with systemic disease. Individuals with systemic disorders such as Down's syndrome, Papillon–Lefèvre syndrome, neutropenia, diabetes, hypophosphatasia, leucocyte adhesion deficiency, histiocytosis X, Chédiak–Higashi syndrome, or those infected with human immunodeficiency virus (HIV) may be predisposed to severe generalized periodontitis. According to the age of onset, the periodontitis may then be classifiable either as generalized prepubertal, generalized juvenile, rapidly progressive or adult. However, in these cases where the periodontitis is identified aetiologically with a systemic disorder, it may be preferable to assign it to a separate category, as illustrated in Table 5.1, rather than include it with forms of periodontitis where the nature of the impaired defence mechanism is uncertain.

Most periodontal lesions, which are associated with an underlying systemic disease, are non-specific. HIV-associated periodontitis, however, may be an exception (*see* Chapter 19): in the later

stages of HIV infection, when the patient's immune system is severely compromised, gingival ulceration with necrosis of underlying soft tissue and alveolar bone may lead to advanced gingival recession and tooth exfoliation. Pain and spontaneous bleeding are common.

On the whole, with the exception of localized juvenile periodontitis, the various forms of chronic periodontitis are poorly characterized. There is considerable overlap and a precise diagnosis is frequently impossible: the time lapse between onset and diagnosis may be unknown; different diagnoses may be appropriate for different teeth in one mouth; and the influence of concurrent systemic disease may be difficult to assess. Failure to make a definitive diagnosis should not, however, prove to be a barrier to therapy, since all forms of periodontitis share a common approach to treatment at the present time. Although adjunctive antimicrobial therapy has been tested in different forms of periodontitis, long-term benefits have not been confirmed.

NON-PLAQUE-ASSOCIATED DISEASES

Non-plaque-associated diseases may occur independently or concurrently with plaque-associated disease. They include traumatic lesions, infections and immunological conditions, as listed in Table 5.1, as well as pathological processes which may affect the periodontal tissue but are not usually considered to be forms of periodontal disease; for example, cysts, neoplasms, renal osteodystrophy, hyperparathyroidism and contact sensitivity reactions. For an extensive review of non-plaque-associated diseases affecting the periodontium, the reader is referred to Newman *et al.* (1993).

OTHER TERMINOLOGY

Refractory periodontitis. This is a term which may be applied to any form of periodontitis, early-onset or adult, which is either unresponsive to treatment or rapidly recurrent in spite of apparently appropriate therapy and good plaque control.

Necrotizing ulcerative periodontitis. This is a term of fairly recent origin, used to describe a situation where periodontitis and acute necrotizing ulcerative gingivitis (ANUG) co-exist, in some

cases because recurrent ANUG has apparently progressed to periodontitis.

Postjuvenile periodontitis. This is a term, now infrequently used, which describes cases of localized juvenile periodontitis which are diagnosed in adulthood and are apparently no longer progressive, being confined to incisors and first molars.

EPIDEMIOLOGY

Epidemiological studies are carried out to determine population trends in the occurrence and distribution of periodontal disease. Although bacterial plaque is clearly the primary aetiological factor in periodontal disease, it is common to find, among *individuals* of the same age and standard of oral hygiene, a significant variation in periodontal disease which may be attributed to the efficacy of their inflammatory and immune mechanisms or to their specific plaque microfloras. Most large populations seem to include a similar mixture of resistant and susceptible individuals so that the susceptibility profile will be relatively constant between different populations. Thus, at a *population* level, when groups of a similar age range are compared, it is apparent that oral hygiene is the principal determinant of periodontal status . The variation in prevalence and severity which exists between different countries or communities, therefore, can be attributed largely to socioeconomic and cultural differences, which are the major determinants of oral hygiene levels. A few populations have been found, however, where this formula does not apply and the amount of periodontal disease is not consistent with plaque levels. Genetic factors within closed communities or severe malnutrition are thought, in these cases, to be critical determinants of periodontal disease susceptibility, confounding the otherwise consistent association between population levels of plaque and periodontal disease.

Prevalence and severity

The term 'prevalence' should serve to indicate, for a given age group, the proportion of the population affected by a given degree of severity of periodontal disease.

Gingivitis. Gingivitis is common in children, and was found in 36% of 3-year olds, 64% of 5-year olds, 97% of 10-year olds and

74% of 15-year olds in Sweden (Hugoson *et al.*, 1981). The high prevalence in 10-year olds may be attributable to deterioration in the gingival condition of exfoliating teeth, and the lowered prevalence at 15 years to the effect of increased social awareness on oral hygiene. There is a further rise in gingivitis prevalence in adulthood.

Periodontitis. In a retrospective bitewing radiographic study of 1026 children and adolescents, from a mainly Hispanic community in the USA, Bimstein *et al.* (1988) found clear evidence of marginal bone destruction (distance between amelocemental junction and alveolar crest >3 mm) in 7.6% of 4-year olds. The peak prevalence (17.9%) occurred among 7-year olds. Between 9 and 14 years, diagnosis became unreliable due to the shedding of deciduous teeth and the eruption of their permanent successors. Among 15–17-year olds, there was a prevalence of 7%.

The true prevalence of periodontitis or marginal bone loss in teenage populations is much disputed, and to a large extent this is due to lack of uniformity in the stringency of diagnostic criteria which are applied. There is wide agreement that large amounts of periodontal destruction are unusual, but minor amounts may be quite common. In a 5-year longitudinal study of 167 British adolescents, initially 14 years of age, from a low socioeconomic area, the prevalence of attachment loss was determined by examining the mesial surfaces of first molars, first premolars and central incisors for evidence of a reduction in attachment level of 1 mm or more (Clerehugh *et al.*, 1990). The prevalence increased from 3% at 14 years, through 37% at 16 years, to 77% at 19 years. Attachment loss greater than 1 mm was found only at 19 years when it affected 14% of the group, and did not exceed 2 mm. The proportion of *sites* with loss of attachment increased from less than 1% at 14 years, through 7% at 16 years, to 31% at 19 years.

In planning an overview of the occurrence and distribution of periodontitis in adults, there is a huge variety of different types of study to choose from. The data for 550 dentate subjects, illustrated in Table 5.2, are from an epidemiological survey in 1983 of 600 randomly selected individuals, evenly distributed into age groups, from a medium-sized town in Sweden (Hugoson *et al.*, 1992). Following a detailed clinical and radiographical examination, these individuals were assigned to one of five periodontal disease groups, the inclusion criteria for which are summarized below:

Group 1 negligible signs of inflammation and no proximal surface bone loss.

Table 5.2. *Number of dentate individuals of each age and the percentage distribution according to severity of periodontal disease (after Hugoson* et al., *1992)*

Age (years)	Number of dentates	Percentages Periodontal disease groups 1	2	3	4	5
20	100	58	40	1	1	
30	98	35	42	21	2	
40	99	17	22	57	3	1
50	99	8	11	70	9	3
60	84	12	4	53	26	6
70	70	1	7	49	38	4
	550	23	22	41	11	2

Group 2 generalized gingivitis but essentially no proximal surface bone loss.

Group 3 generalized early bone loss (less than a third of normal bone height).

Group 4 generalized moderate bone loss (one-third to two-thirds of normal bone height).

Group 5 generalized advanced bone loss (more than two-thirds of normal bone height).

Table 5.2 shows that, out of the entire sample, 23% were essentially free of periodontal disease (group 1), while a further 22% had gingivitis without significant evidence of bone loss (group 2). The commonest diagnosis was generalized early marginal bone loss (group 3), which affected 41%, while only 11% and 2% of the entire sample suffered from generalized moderate (group 4) and advanced bone loss (group 5), respectively. The prevalence and severity of bone loss increased with increasing age. However, generalized advanced bone loss was not diagnosed before age 40 years and was uncommon at all ages thereafter. Generalized moderate bone loss affected only 1% of 20-year olds and 2% of 30-year olds, perhaps reflecting the prevalence of so-called rapidly progressive periodontitis. On the other hand, generalized moderate bone loss was common at older age levels affecting 26% of 60-year olds and 38% of 70-year olds.

Although 55% of the entire population were affected by marginal bone loss which, of course, is irreversible, many had already received periodontal treatment. Thus, when gingival bleeding and

Table 5.3. *The proportions of different populations with periodontal destruction equivalent to at least one-third of root length affecting at least one tooth surface*

Country	Number in age group	Age group (years)	Disease level	Prevalence (%)
Japan*	51	30–39	≥ 5 mm loss of attachment	31
Sweden†	52	35	≥ 6 mm bone level reduction	38
Scotland‡	67	35-39	> 50% bone loss	27

* Okamoto *et al.*, 1988.
† Papapanou *et al.*, 1988.
‡ Jenkins and Kinane, 1990.

probing depth data were assessed in conjunction with marginal bone levels, approximately one-third of the 55% with marginal bone loss were essentially periodontally healthy, although with reduced periodontal support.

In extrapolating the results of this Swedish study to other parts of the world, it must be remembered that these data were collected from a population with a high level of dental awareness. Nevertheless, there is good agreement with cross-sectional studies of various other adult populations that *generalized* moderate or advanced bone loss is confined to 10–15% of the population.

The grouping of patients into one of five categories, above, according to their 'whole mouth scores', results in considerable smoothing of data and disguises the prevalence of different threshold levels of disease, i.e. the proportion of patients with at least one tooth surface affected beyond certain disease thresholds. For example, in this study, only 2% of 30-year olds and 4% of 40 year-olds had *generalized* bone loss exceeding one-third of average root length. Table 5.3, however, shows that, in three other studies of different populations, the reported prevalence of attachment loss or bone loss, exceeding one-third of average root length, and affecting at least one tooth surface, was 27–38% of individuals in the 30–39-year age range.

Finally, caution must be exercised in interpreting the results of all these studies since a much higher prevalence of periodontitis in older individuals could be overlooked by failure to account for the periodontal conditions of missing teeth.

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